COLL·MED·PHILAD·INSTIT·A·D·MDCCLXXXVII·

Digitized by the Internet Archive
in 2013

http://archive.org/details/transactionsofco3521coll

TRANSACTIONS

OF THE

COLLEGE OF PHYSICIANS

OF

PHILADELPHIA

THIRD SERIES

VOLUME THE FIFTY-SECOND

PHILADELPHIA
PRINTED FOR THE COLLEGE
1930

NOTICE

The present volume of TRANSACTIONS contains the papers read before the College from January, 1930, to December, 1930, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its TRANSACTIONS.

EDITED BY

WALTER G. ELMER, M.D.

"This volume is published in part by the income from the Francis Houston Wyeth Fund."

DORNAN, PRINTER

CONTENTS

List of Presidents of the College vii
List of Officers and Standing Committees viii
List of Fellows of the College xi
List of Associate Fellows of the College lvii
Necrological List for 1930 lix
List of Prizes and Lectures lx
Memoir of Charles Eucharist de Medicis Sajous, M.D., Sc.D., LL.D. . lxv
Memoir of Thomas H. Fenton, M.D. lxxi
Memoir of Henry Morris, M.D. lxxiii

Annual Address of the President. By John H. Gibbon, M.D. . . . 1
The Mütter Museum of the College of Physicians of Philadelphia. By Harry P. Schenck, M.D. 6
Goiter in the Philadelphia District. By Edward Rose, M.D., and Charles H. Frazier, M.D. 12
Congenital Insufficiency of the Palate. By George M. Dorrance, M.D. 19
Additional Observations on the Effect of Sodium Amytal Anesthesia Upon the Uterus and Its Use in Obstetrics and Gynecology. By John C. Hirst, B.A., M.D., and Maxwell E. Lapham, M.D. . . 47
The Relationship Between Inorganic Dust and Pulmonary Disease. By A. J. Lanza, M.D. 55
Safe Limits of Atmospheric Dustiness and How They Are Measured. By Henry Field Smyth, M.D., Dr. P.H. 66
Medicine in the Sub-arctic. The Mary Scott Newbold Lecture. Lecture XXII. By Sir Wilfred Grenfell, K.C.M.G., M.D. 73
Psychosis, Neurosis and Character-Disturbance. The Nathan Lewis Hatfield Lecture. Lecture X. By Franz Alexander, M.D. . . 96
Regeneration Reaction to Sulfhydryl and Its Partially Oxidized Derivatives. By Frederick S. Hammett, Ph.D. 108
Hepatitis. By John B. Deaver, M.D. 110
Bacteriology, Etiology and Serotherapy of Infections of Wounds (Traumatoses), Especially of Gas Gangrene. The Mary Scott Newbold Lecture. Lecture XXIV. By Prof. M. Weinberg 118
The Management of Goiter and Hyperthyroidism. The Mütter Lecture. By Frank H. Lahey, M.D. 133
Galen. By Charles W. Burr, M.D. 155

Galen's Ophthalmology. By Burton Chance, M.D. 166
The Origin of the University of Bologna (Italy). By Giuseppe Franchini, M.D. 173

Proceedings of the Section on Ophthalmology

Iridectomy in Bullous Keratitis. By William Zentmayer, M.D. . . . 187
A Case of Electric Cataract. By T. B. Holloway, M.D. 189
Adenocarcinoma of Conjunctiva. By Edward B. Spaeth, M.D. . . 189
A Method of Uncovering Latent Hyperphoria. By Leighton F. Appleman, M.D. 190
Skin Grafting of Eyelids. By Burton Chance, M.D. 193
Glioma. By Burton Chance, M.D. 194
Cholesterin in the Anterior Chamber. By Leighton F. Appleman, M.D. 194
Exhibition of Case of Heterochromic Cyclitis. By Warren S. Reese, M.D. 196
Hole at the Macula. By George H. Cross, M.D. 198
Treatment of Vernal Conjunctivitis by Radium. By Ronald C. Moore, M.D. 200
Ephedrine and Glaucoma. By A. Barlow, M.D. 203
Differential Diagnosis Between Tumor and Choroidal Detachment. By George Schwartzkopf, M.D. 204
Tendon Transplantation for Paralysis of the Superior Rectus. By Luther C. Peter, M.D. 205
Foreign Body in the Lens. By George Cross, M.D. 207
Variations of the Intraneural Course of the Central Vein of the Retina. By W. E. Fry, M.D. 207
Case of Acute Vitreous Haze Following the External Use of Adrenalin and Scopolamin for the Separation of Synechiæ. By H. Maxwell Langdon, M.D. 209
Some Interesting Cases of Sclerocorneal Trephining in Chronic Simple Glaucoma. (A Clinical Paper.) By William Zentmayer, M.D. . 210
The Treatment of Argyrosis. Exhibition of Case. By I. S. Tassman, M.D. 212
Ophthalmic Operations. By Frank C. Parker, M.D. 213
Congenital Total Color-blindness. By W. E. Fry, M.D. 213
Résumé of the Special Inquiry of Eye Health in Philadelphia. By Haven Emerson, M.D. 215
Emergency Outfit for Ophthalmic Examination and Treatment. By Edward A. Shumway, M.D. 217
Trachoma in Wrestlers. By William Zentmayer, M.D. 217

Incorporation in the Prescription of the Style and Shape of the Lenses. By Alfred Cowan, M.D. 219

Modified Le Grange Operation for Glaucoma. By J. Milton Griscom, M.D. 221

The Bacteriologic and Immunologic Aspects of Iritis, With Special Reference to Its Relationship to Bacterial Allergy. By John A. Kolmer, M.D. 223

Roentgentherapy in Pituitary Tumors. By George E. Pfahler, M.D. and E. W. Spachman, M.D. 225

The Surgical Treatment of Pituitary Tumors. By Charles H. Frazier, M.D., Sc.D. 227

The Ophthalmological Aspects of Pituitary Tumors. By T. B. Holloway, M.D. 229

Proceedings of the Section on Otology and Laryngology

Observations on the Transillumination of the Frontal Sinuses. By Edward H. Campbell, M.D. 231

Some Practical Points in the Use of Electrosurgical Measures in the Upper Respiratory Tract. By Marshall B. Sponsler, M.D. . . 233

Two Cases of Brain Abscess Presenting Some Interesting Features. By Austin T. Smith, M.D. 235

Vasomotor Rhinitis. By Harold G. Tobey, M.D. 238

Symposium on the Relation of Sinus Disease to Pulmonary Infection, from the Standpoint of the Internist. By Thomas McCrae, M.D. . . 243

From the Standpoint of the Rhinologist. By William V. Mullin, M.D 245

From the Standpoint of the Bronchoscopist. By Robert M. Lukens, M.D. 247

From the Standpoint of the Roentgenologist. By Willis F. Manges, M.D. 249

Some Notes on Agranulocytic Angina: Report of Case With Apparent Recovery. By James A. Babbitt, M.D., and Thomas Fitz-Hugh, Jr., M.D. 251

Bursa Pharyngea. (Lantern Demonstration.) By George M. Dorrance, M.D. 254

Regeneration of the Mucous Membrane of the Frontal Sinuses. By George M. Coates, M.D., and Matthew S. Ersner, M.D. . . 256

Presentation of a New Type of Tracheotomy Tube With Valve. By Gabriel Tucker, M.D. 257

The Present Status of the Intracarotid Method of Treatment of Pneumococcus and Streptococcus Meningitis. By John A. Kolmer, M.D. 258

The Specific Prophylaxis of Pneumococcus and Streptococcus Meningitis. By John A. Kolmer, M.D., and Dr. K. W. Amano. 259

Treatment of Stricture of the Oral Pharynx. By George M. Dorrance, M.D. 263

Orbital Abscess Following Sinus Disease. By John R. Davies, Jr., M.D. 264
Acute Mastoiditis With Basilar Meningitis. By William Hewson, M.D. 265
Compound Fracture of the Malar Bone With Involvement of the Maxillary Sinus. By Thomas G. Aiken, M.D. 267
Chrondromata of the Larynx. Report of a Case. By Robert M. Lukens, M.D. 268

Proceedings of the Section on General Medicine 270
Proceedings of the Section on Public Health and Industrial Medicine 274
Proceedings of the Section on Medical History 275

Annual Report of the Library Committee 277

LIST

OF THE

PRESIDENTS OF THE COLLEGE FROM THE TIME OF ITS INSTITUTION

ELECTED	
1787	JOHN REDMAN
1805	WILLIAM SHIPPEN
1809	ADAM KUHN
1818	THOMAS PARKE
1835	THOMAS C. JAMES*
1835	THOMAS T. HEWSON
1848	GEORGE B. WOOD
1879	W. S. W. RUSCHENBERGER
1883	ALFRED STILLÉ
1884	SAMUEL LEWIS†
1884	J. M. DA COSTA
1886	S. WEIR MITCHELL
1889	D. HAYES AGNEW
1892	S. WEIR MITCHELL
1895	J. M. DA COSTA
1898	JOHN ASHHURST, JR.
1900	W. W. KEEN
1902	HORATIO C. WOOD
1904	ARTHUR V. MEIGS
1907	JAMES TYSON
1910	GEORGE E. DE SCHWEINITZ
1913	JAMES CORNELIUS WILSON
1916	RICHARD H. HARTE
1919	WILLIAM J. TAYLOR
1922	THOMAS R. NEILSON
1925	HOBART A. HARE
1928	JOHN H. GIBBON

* Died four months after his election.
† Resigned on account of ill-health.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

1930

OFFICERS AND STANDING COMMITTEES

PRESIDENT

JOHN H. GIBBON, M.D.

VICE-PRESIDENT

FRANCIS R. PACKARD, M.D.

CENSORS

GEORGE E. DE SCHWEINITZ, M.D.
JAMES C. WILSON, M.D.
WILLIAM J. TALYOR, M.D.
THOMAS R. NEILSON, M.D.

SECRETARY

JOHN H. GIRVIN, M.D.

TREASURER

CHARLES F. MITCHELL, M.D.

HONORARY LIBRARIAN

ASTLEY P. C. ASHHURST, M.D.

COUNCILLORS

To serve until January, 1931

WILLIAM R. NICHOLSON, M.D.
GEORGE M. COATES, M.D.

To serve until January, 1932

JOHN B. CARSON, M.D.
EDWARD B. HODGE, M.D.

To serve until January, 1933

WILLIAM D. STROUD, M.D.
HERMAN B. ALLYN, M.D.

COMMITTEE OF PUBLICATION

THOMPSON S. WESTCOTT, M.D., *Ch'n*
WALTER G. ELMER, M.D.
ARTHUR H. HOPKINS, M.D.

LIBRARY COMMITTEE

GEORGE W. NORRIS, M.D., *Ch'n*
ALBERT P. BRUBAKER, M.D.
EDWARD B. KRUMBHAAR, M.D.
JOHN H. JOPSON, M.D.
H. R. M. LANDIS, M.D.
The HONORARY LIBRARIAN *ex-officio*

COMMITTEE OF MÜTTER MUSEUM

GEORGE P. MÜLLER, M.D., *Ch'n*
MILTON J. GREENMAN, M.D.
HERBERT FOX, M.D.

HALL COMMITTEE

E. HOLLINGSWORTH SITER, M.D., *Ch'n*
B. ALEX. RANDALL, M.D.
ELLISTON J. MORRIS, M.D.
GEORGE W. OUTERBRIDGE, M.D.
PHILIP F. WILLIAMS, M.D.

COMMITTEE ON DIRECTORY FOR NURSES

Frederick Fraley, M.D., *Ch'n* — Horace H. Jenks, M.D.
Henry P. Brown, Jr., M.D.

COMMITTEE ON FINANCE

William J. Taylor, M.D., *Ch'n* — O. H. P. Pepper, M.D.
David Riesman, M.D. — The President and Treasurer, *ex-officio*

COMMITTEE ON ENTERTAINMENTS

Norris W. Vaux, M.D., *Ch'n* — Henry P. Brown, Jr., M.D.
Emory G. Alexander, M.D. — Arthur Newlin, M.D.
The President, *ex-officio*

COMMITTEE ON SCIENTIFIC BUSINESS

W. Estell Lee, M.D., *Ch'n* — Henry K. Mohler, M.D.
Damon B. Pfeiffer, M.D. — The President and Secretary, *ex-officio*

COMMITTEE ON PUBLIC HEALTH AND PREVENTIVE MEDICINE

Elmer H. Funk, M.D., *Ch'n* — James M. Anders, M.D.
Henry Field Smyth, M.D. — The President, *ex-officio*

COMMITTEE ON ALVARENGA PRIZE

Philip F. Williams, M.D., *Ch'n* — Richard A. Kern, M.D.
Edward B. Hodge, M.D. — B. F. Crawford, M.D.
William D. Stroud, M.D.

COMMITTEE ON NATHAN LEWIS HATFIELD PRIZE AND LECTURESHIP

(until February, 1931)

George M. Piersol, M.D., *Ch'n* — Edward A. Strecker, M.D.
Robert G. Torrey, M.D.

TRUSTEES OF NATHAN LEWIS HATFIELD PRIZE AND LECTURESHIP

R. N. Downs, Jr., M.D. — Henry K. Dillard, Jr., M.D.
Arthur H. Gerhard, M.D.

TRUSTEE UNDER THE WILL OF THOMAS SKELTON HARRISON

(until February, 1932)

Frederick Fraley, M.D.

COMMITTEE ON WEIR MITCHELL ORATION

(until February, 1933)

G. E. de Schweinitz, M.D., *Ch'n* — Francis W. Sinkler, M.D.
Charles H. Frazier, M.D. — Albert P. Brubaker, M.D.
Charles W. Burr, M.D.

COMMITTEE ON MARY SCOTT NEWBOLD LECTURES

William J. Taylor, M.D., *Ch'n* — Williams B. Cadwalader, M.D.
Edward J. Klopp, M.D.
The President and Chairman Committee on Scientific Business, *ex-officio*

SECTIONS

Ophthalmology—*Chairman*, H. Maxwell Langdon, M.D.
Clerk, A. G. Fewell, M.D.

Otolaryngology—*Chairman*, George M. Coates, M.D.
Clerk, Frederick M. Strouse, M.D.

General Medicine—*Chairman*, Truman G. Schnabel, M.D.
Clerk, Henry K. Mohler, M.D.

Medical History—*Chairman*, Herman B. Allyn, M.D.
Clerk, E. J. G. Beardsley, M.D.

Public Health and Industrial Medicine—*Chairman*, D. H. Bergey, M.D.
Clerk, Alex. Heron Davisson, M.D.

FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA

DECEMBER, 1930

* Non-resident Fellows. † Fellows who have commuted dues. ‡ Deceased.

ELECTED

1892. Abbott, Alexander Crever, M.D., Sc.D., Dr. P.H., Professor of Hygiene and Bacteriology and Director of the School of Hygiene and Public Health, University of Pennsylvania. 4016 Pine St.

1912. Addison, William H. F., A.B., M.D., Professor of Normal Histology and Embryology in the University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1926. Adler, Francis Heed, M.A., M.D., Assistant Surgeon, Wills Eye Hospital; Instructor in Ophthalmology and Physiology, University of Pennsylvania. 313 S. Seventeenth St.

1905. Adler, Lewis H., Jr., M.D., Formerly Professor of Proctology in the Graduate School of Medicine, University of Pennsylvania; formerly Prosector to the Professor of Anatomy in the University of Pennsylvania; Consulting Surgeon to the Charity Hospital. 313 S. Seventeenth St.

1914. Aiken, Thomas Gerald, M.D., Physician-in-Chief to the Chester County Hospital, West Chester, Pa.; Pathologist to the Country Branch of the Rush Hospital for Tuberculosis. Berwyn, Pa.

‡1913. Alexander, Emory G., M.D., Assistant Professor of Surgery, Medical School, University of Pennsylvania; Surgeon to the Episcopal, St. Christopher's Hospital for Children and Abington Hospitals. 337 S. Eighteenth St.

1906. Allen, Francis Olcott, Jr., A.B., M.D. 2216 Walnut St.

1896. Allyn, Herman B., A.B., M.D., Consulting Physician to the Philadelphia General Hospital. 3910 Chestnut St.

1888. Anders, James M., M.D., LL.D., Sc.D., Professor of Medicine in the Graduate School of Medicine, University of Pennsylvania; Officer de l'Instruction Publique. 250 S. Seventeenth Street.

ELECTED

1905. Anspach, Brooke M., M.D., Professor of Gynecology in the Jefferson Medical College; Attending Gynecologist, Jefferson Hospital; Attending Gynecologist to the Bryn Mawr Hospital. 1827 Spruce St.

1930. Appel, Kenneth E., A.B., A.M., Ph.D., M.D., Assistant Professor of Psychiatry, Medical School, University of Pennsylvania; Chief of Out-Patient Department and Psychiatrist, Institute for Mental Hygiene, Pennsylvania Hospital; Chief of Neuro-Psychiatric Pay Clinic, Pennsylvania Hospital, Haverford, Pa.

1905. Appleman, Leighton F., M.D., Attending Surgeon to the Wills Eye Hospital; Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Ophthalmologist to the Frederick Douglass Memorial Hospital and to the Burd School; Demonstrator of Pharmacy and Materia Medica, and Instructor in Therapeutics in the Jefferson Medical College. 308 S. Sixteenth St.

1922. Arnett, John Hancock, A.B., M.D., Physician to the Episcopal Hospital; Instructor in Medicine, Medical School, University of Pennsylvania. 2116 Pine St.

1906. Ashhurst, Astley Paston Cooper, A.B., M.D., Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the Episcopal Hospital and to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases. 257 S. Sixteenth St.

1893. Ashton, Thomas G., M.D. Wynnewood, Pa.

1920. Astley, George Mason, M.D., Assistant Surgeon, Samaritan Hospital. 5317 Master St.

1914. Austin, J. Harold, B.S. (Univ. of Penna.), M.D., Professor of Research Medicine, University of Pennsylvania. John Herr Musser Department of Research Medicine, University of Pennslyvania.

1906. Babbitt, James A., A.B. (Yale), A.M. (Haverford), M.D., Assistant Professor of Otolaryngology, University of Pennsylvania; Associate Professor of Otolaryngology, Graduate School of Medicine, University of Pennsylvania; Laryngologist and Aurist, Lankenau Hospital; Otolaryngologist, Children's Hospital. 1912 Spruce St.

1910. Baer, Benjamin F., Jr., B.S., M.D., Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Assistant Ophthalmologist, University Hospital; Attending Surgeon to Wills Eye Hospital; Ophthalmologist, Howard Hospital. 1927 Spruce St.

ELECTED

1911. Baldwin, James Harvey, A.B., M.D., Surgeon to the Methodist Hospital. 1426 Pine St.

1916. Balentine, Percival L., M.D., Assistant Surgeon, Wills Eye Hospital. 302 Weightman Building, 1524 Chestnut St.

1898. Balliet, Tilghman M., A.M., M.D., Professor of Therapeutics at Dartmouth College, Hanover, N. H.; Physician to the Old Man's Home. 3920 Sansom St.

1911. Barnard, Everett P., M.D., Obstetrician to the Maternity Hospital; Assistant Instructor in Obstetrics in the University of Pennsylvania. 1820 S. Rittenhouse Square.

1924. Bartle, Henry J., B. S., M.D., Clinical Assistant, Department for Disease of the Stomach and Bowels, Jefferson Medical College and Hospital. 1930 Chestnut St.

1921. Bauer, Edward L., M.D., Professor of Children's Diseases, Jefferson Medical College; Pediatrist to the Jefferson and Memorial Hospitals and the Philadelphia Hospital for Contagious Diseases; Consulting Pediatrist, Germantown Hospital. 345 S. Nineteenth St.

1883. Baum, Charles, A.M., M.D., Ph.D. 1828 Wallace St.

1922. Bazett, Henry Cuthbert, M.A., M.D. (Oxon.), F.R.C.S. (England), Professor of Physiology, University of Pennsylvania. Bryn Mawr, Pa.

1908. Beardsley, Edward J. G., M.D., L.R.C.P. (Lond.), Clinical Professor of Medicine in the Jefferson Medical College; Chief Clinical Assistant in the Out-Patient Medical Department of the Jefferson Medical College Hospital; Assistant Physician to the Jefferson Hospital. 1919 Spruce St.

1930. Beardwood, Joseph T., A.B., M.D., Associate Physician, Presbyterian Hospital; Associate in Cardiology, Graduate School of Medicine, University of Pennsylvania. 256 S. Twenty-first St.

1930. Belk, William Parks, A.B., M.D., Pathologist, Bryn Mawr Hospital; Senior Histologist, Philadelphia General Hospital. Bryn Mawr, Pa.

*1916. Bell, William Hemphill, M.D., Captain, Medical Corps, U. S. Navy. 1869 Wyoming Ave., N.W., Washington, D. C.

1923. Bergey, David H., A.M., M.D., B.S., Dr. P.H., Professor of Hygiene and Bacteriology, University of Pennsylvania. 206 S. Fifty-third St.

*1903. Biggs, Montgomery H., M.D., Surgeon to the Rutherford Hospital; Chief Surgeon of the Carolina, Churchfield and Ohio Railway. Rutherfordton, N. C.

ELECTED

1918. Billings, Arthur E., M.D., Assistant Professor of Surgery, Jefferson Medical College; Assistant Surgeon, Jefferson Hospital; Surgeon to Pennsylvania and Bryn Mawr Hospitals. 2020 Spruce St.

1917. Birdsall, Joseph C., A.B., A.M., M.D., Associate Professor of Urology in the Graduate School of Medicine, and Urologist to the Hospital of the Graduate School of Medicine, University of Pennsylvania; Assistant in Genito-urinary Dispensary, Presbyterian Hospital. 4610 Spruce St.

1908. Bland, Pascal Brooke, M.D., Professor of Obstetrics, Jefferson Medical College; Visiting Obstetrician, Jefferson Medical College; Gynecologist to St. Joseph's Hospital. 1621 Spruce Street.

1917. Block, Frank B., A.B., M.D., Surgeon to the Jewish Hospital; Associate in Gynecology in the University of Pennsylvania. Medical Arts Building.

1894. Bochroch, Max H., M.D., Professor of Nervous and Mental Diseases, Temple University; Visiting Physician to the Psychopathic Wards of the Philadelphia General Hospital. 1539 Pine St.

1929. Bockus, Harry L., B.S., M.D., Professor of Gastroenterology, Graduate School of Medicine, University of Pennsylvania, and Attending Gastroenterologist, Graduate Hospital; Attending Physician, Presbyterian Hospital. 250 S. Eighteenth St.

1896. Boger, John A., A.M., M.D., Surgeon to St. Mary's Hospital; Surgeon to the Stetson Hospital. 2213 N. Broad St.

1928. Boles, Russell S., M.D., Associate in Medicine, University of Pennsylvania; Visiting Physician, Philadelphia General Hospital; Consulting Gastroenterologist, U. S. Veterans' Bureau, Philadelphia. Rittenhouse Plaza.

1923. Bond Earl, D., A.B., M.D., Physician-in-Chief, Pennsylvania Hospital, Department for Mental and Nervous Diseases; Professor of Psychiatry, School of Medicine and Graduate School of Medicine, University of Pennsylvania. Bryn Mawr, Pa.

1911. Bonney, Charles W., A.B., M.D., Assistant Professor of Topographic and Applied Anatomy in the Jefferson Medical College. 1117 Spruce St.

1924. Boon, David J., M.D., Opthalmologist, Memorial Hospital and Philadelphia Hospital for Contagious Diseases; Assistant Ophthalmic Surgeon, Episcopal Hospital. 1532 N. Fifteenth Street.

ELECTED

1921. Borzell, Francis Frank, M.D., Roentgenologist, Frankford Hospital; Chief Clinical Assistant, Roentgen Department, Jefferson Medical College; *X*-Ray Consultant, State Hospital for the Insane, Norristown, Pa. 4910 Frankford Ave.

1913. Boston, L. Napoleon, A.M., M.D., Professor of Physical Diagnosis, Graduate School of Medicine, University of Pennsylvania; Professor of Principles and Practice of Medicine and Clinical Medicine, Woman's Medical College. 1930 Chestnut St.

1928. Bothe, Albert Edward, B.S., M.D., M.S., Instructor in Surgery, Medical School and Instructor in Urology, Graduate School, University of Pennsylvania; Assistant in Urology, Presbyterian Hospital; Urologist, Misericordia Hospital. 133 S. Thirty-sixth St.

1928. Bothe, Frederick A., M.S., M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Assistant Surgeon, Presbyterian Hospital; Associate Surgeon, Children's Hospital. 133 S. Thirty-sixth St.

1919. Bowen, David Ralph, M.D., Radiologist to the Pennsylvania Hospital. 82 W. Lacrosse Ave., Lansdowne, Pa.

1923. Bower, John O., M.D., Surgeon to the Philadelphia General and United States Veterans' Hospitals; Clinical Professor of Surgical Research, Temple University. 2008 Walnut St.

1891. Boyd, George, M., M.D., Professor of Obstetrics, Graduate School of Medicine, University of Pennsylvania; Consulting Obstetrician to the Preston Retreat and Philadelphia General Hospital. 1909 Spruce St.

1907. Boyer, Henry Percival, M.D., Neurologist to the Stetson Hospital; Physician to the Philadelphia Home for Incurables; Medical Electrician to the Orthopedic Hospital and Infirmary for Nervous Diseases. 4602 Baltimore Ave.

1925. Boykin, Irvine M., M.D., Associate Surgeon to the Episcopal Hospital; Associate in Surgery, Graduate School of Medicine, Instructor in Surgery, School of Medicine, University of Pennsylvania. 922 Medical Arts Building.

1929. Bradbury, Samuel, M.D., Medical Director, Out-Patient Department, Pennsylvania Hospital. 151 W. Coulter St.

1907. Bradley, William N., Ph.G., M.D., Visiting Pediatrist to Philadelphia General Hospital; Associate in Pediatrics, School of Medicine, University of Pennsylvania; Associate Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania. 1725 Pine St.

ELECTED

1921. Bransfield, John W., M.D., Clinical Professor of Surgery, Woman's Medical College; Visiting Surgeon to St. Agnes, St. Vincent's and Woman's College Hospitals. 2101 Spruce Street.

1903. Branson, Thomas F., M.D., Attending Physician to the Bryn Mawr Hospital. Rosemont, Pa.

1900. Brinton, Ward, A.M., M.D., Chief of Division of Tuberculosis, Department of Public Health of Philadelphia; Clinical Professor of Medicine, Woman's Medical College of Pennsylvania; Visiting Physician, Department for Tuberculosis, Philadelphia General Hospital, and Eaglesmere Sanatorium. 1423 Spruce St.

1917. Bromer, Ralph S., A.B. (Yale), M.D., Associate in Radiology, Graduate School of Medicine, and Instructor in Roentgenology, School of Medicine, University of Pennslyvania; Roentgenologist to the Episcopal, the Orthopedic and the Children's Hospitals. 504 W. Mt. Airy Ave.

1919. Brown, Henry P. Jr., M.D., Surgeon and Chief of Out-Patient Clinic of the Pennsylvania Hospital; Surgeon to the Children's Hospital; Associate Surgeon, Presbyterian Hospital; Instructor in Surgery in the School of Medicine, and Assistant Professor of Surgery in the Graduate School of Medicine, University of Pennsylvania. 624 Medical Arts Building.

1916. Brown, Samuel Horton, M.D. 1930 Chestnut St.

1887. Brubaker, Albert P., A.M., M.D., Emeritus Professor of Physiology and Medical Jurisprudence in the Jefferson Medical College. 3426 Powelton Ave.

1930. Bucher, Carl J., B.S., M.D. The Westbury, Fifteenth and Spruce Sts.

1916. Buckley, Albert Coulson, M.D., Medical Superintendent, Friends' Hospital, Frankford; Associate Professor of Psychiatry, Graduate School of Medicine, University of Pennsylvania. Frankford, Phila.

1928. Burden, Verne G., M.D., M.S., Surgeon to St. Joseph's and Philadelphia General Hospitals; Instructor in Surgery, Graduate School of Medicine, University of Pennsylvania. The Warwick, Seventeenth and Locust Sts.

1927. Burns, Michael A., M.D., Assistant Professor Nervous and Mental Diseases, Jefferson Medical College; Chief of the Nervous and Mental Clinic, Jefferson Hospital; Visiting Neurologist, Philadelphia General Hospital; Neuropsychiatrist, St. Mary's Hospital. N. E. Cor. Twentieth and Chestnut Sts.

ELECTED

1906. Burns, Stillwell C., M.D., Associate Professor of Surgery in the Graduate School of Medicine, University of Pennsylvania. 1925 Spring Garden St.

1892. Burr, Charles W., M.D., Professor of Mental Diseases in the University of Pennsylvania; Neurologist to the Philadelphia General Hospital. 1918 Spruce St.

1906. Butler, Ralph, M.D., Professor of Laryngology in the Graduate School of Medicine, University of Pennsylvania; Laryngologist and Aurist to the Lankenau Hospital. 1930 Chestnut Street.

*1908. Cadbury, William W., A.M., M.D., Lingnan University, Canton, China.

†1907. Cadwalader, Williams B., M.D., Clinical Professor of Neurology, School of Medicine, University of Pennsylvania; Neurologist to the Presbyterian Hospital; Consulting Neurologist to the Bryn Mawr Hospital. 133 S. Thirty-sixth St.

1928. Campbell, Edward H., M.D., Assistant Otolaryngologist, Pennsylvania and Children's Hospitals, and in Out-Patient Department of Lankenau Hospital; Instructor in Otolaryngology in the Medical School and Graduate School of Medicine, University of Pennsylvania. 2117 Chestnut St.

1905. Carmany, Harry S., M.D., Surgeon to Memorial Hospital, Roxborough; Associate Surgeon of the Episcopal Hospital. 366 Green Lane, Roxborough.

1910. Carnett, John Berton, M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate in Surgery, Medical Department, University of Pennsylvania; Surgeon to Philadelphia General Hospital; Chief Surgeon to the American Stomach Hospital. 2012 Spruce St.

1930. Carpenter, Chapin, A.B., M.D., Assistant Ophthalmologist, Bryn Mawr Hospital; Surgeon, Eye Dispensary, University Hospital; Instructor in Ophthalmology, Under-Graduate Department, University of Pennsylvania. 1930 Chestnut St.

1905. Carpenter, Herbert B., M.D. 1805 Spruce St.

1926. Carpenter, Howard Childs, M.D., Vice-Dean for Pediatrics and Professor of Pediatric Hygiene, Graduate School of Medicine, University of Pennsylvania; Assistant Professor of Pediatrics, School of Medicine, University of Pennsylvania; Director, Department for the Prevention of Disease, Children's Hospital. 1805 Spruce St.

1895. Carpenter, John T., M.D., Consulting Ophthalmologist to the Bryn Mawr and Abington Hospitals. 1930 Chestnut St.

ELECTED

1917. Carson, John B., M.D., Physician to Episcopal Hospital; Assistant Physician, Pennsylvania Hospital. 1802 Pine Street.

1922. Case, Eugene A., M.D., Professor of Morbid Anatomy, Graduate School of Medicine, University of Pennsylvania; Assistant Pathologist, Philadelphia General Hospital. Lansdowne, Pa.

1892. Cattell, Henry W., A.M., M.D., Editor of the International Clinics. Jacksonville Road, Burlington, N. J.

1900. Chance, Burton, M.D., Attending Surgeon to the Wills Eye Hospital; Consulting Ophthalmologist Eastern Pennsylvania Institution for the Feeble-minded and Epileptics; Ophthalmic Surgeon to the Pennsylvania Railroad Company. 315 S. Fifteenth St.

‡1897. Cheston, Radcliffe, M.D., Visiting Physician to the Chestnut Hill Hospital; Consulting Physician to the Germantown Hospital and the the Pennsylvania Institution for the Deaf and Dumb. Chestnut Hill.

1903. Chrystie, Walter, M.D. One of the Senior Physicians to Bryn Mawr Hospital. Bryn Mawr, Pa.

1929. Clark, Eliot R., A.B., M.D., Professor of Anatomy and Director of the Department of Anatomy, School of Medicine, University of Pennsylvania. 301 St. Mark's Square.

1926. Clarke, J. Alexander, A.B., M.D., Chief Clinical Assistant, Out-Patient Department of Applied Immunology, Jefferson Hospital; Assistant Physician, Philadelphia General Hospital; Instructor in Medicine, Undergraduate School, University of Pennsylvania. 334 S. Twenty-first St.

1926. Clerf, Louis H., M.D., Instructor in Bronchoscopy and Esophagoscopy and Demonstrator of Laryngology, Jefferson Medical College; Assistant Professor of Bronchoscopy and Esophagoscopy, Graduate School of Medicine, University of Pennsylvania; Chief Clinical Assistant in Bronchoscopy and Esophagoscopy, Jefferson Hospital; Assistant in Bronchoscopy and Esophagoscopy, University Hospital. 1216 Walnut Street.

1910. Cloud, J. Howard, M.D., Attending Physician to the Bryn Mawr Hospital. 11 W. Montgomery Ave., Ardmore, Pa.

1903. Coates, George Morrison, A.B., M.D., Professor of Otology, Graduate School of Medicine, University of Pennsylvania; Associate Laryngologist, Pennsylvania Hospital; Consulting Otolaryngologist to the Philadelphia Orphanage, U. S. Veterans' Bureau and Base Hospital No. 49, Chester County

ELECTED

Hospital, Lawrenceville School and to the Sharon Hospital, Sharon, Conn. 1721 Pine St.

1908. Codman, Charles A. E., M.D., Physician to the American Oncologic Hospital. 4116 Spruce St.

1922. Cohen, Leon Solis, A.B., M.D., Chief Clinical Assistant of Roentgenology, Jefferson Medical College; Radiologist to the Memorial Hospital, Roxborough; Radiologist and Director of the Research Tumor Clinic, Jewish Hospital. 1923 Spruce St.

1907. Cohen, Myer Solis, A.B., M.D., Assistant Professor of Medicine, Graduate School of Medicine, University of Pennsylvania; Lecturer on Medical Jurisprudence and Toxicology, Woman's Medical College of Pennsylvania; Pediatrist to the Jewish and Mount. Sinai Hospitals. 2029 Spruce St.

1888. Cohen, Solomon Solis, M.D., H.L.D., Emeritus Professor of Clinical Medicine in the Jefferson Medical College; Emeritus Physician to the Jefferson Hospital; Consulting Physician to the Philadelphia General and Jewish Hospitals. 1906 Walnut Street.

1920. Cole, Charles J., A.B., M.D., Visiting Physician, Philadelphia Hospital for Contagious Diseases; Physician to Out-Patient Department, Mary Drexel Home. Elkins Park, Pa.

1898. Coles, Stricker, M.D. Bryn Mawr, Pa.

*1901. Coley, Thomas Luther, A.B., M.D., Major, Medical Corps, U. S. Army. Fort Davis, Canal Zone.

1930. Cooper, David A., B.S., M.D., Instructor in Medicine, University of Pennsylvania. 1520 Spruce St.

1912. Copp, Owen, A.B., M.D., Consultant for the Future Development of the Pennsylvania Hospital. 4401 Market St.

1911. Cornell, Walter Stewart, A.B., M.D., Director of Medical Inspection of Public Schools of the City of Philadelphia; Chief of Medical Staff of the House of Detention; Lecturer on Osteology in the University of Pennsylvania. 1919 Cherry Street.

1914. Corson, Edward Foulke, M.D., Assistant Professor of Dermatology, Jefferson Medical College; Dermatologist, Chilren's and Chestnut Hill Hospitals. Medical Arts Building.

1930. Cottrell, James E., M.D., Instructor in Medicine, Medical School, University of Pennsylvania; Assistant Physician, University Hospital; Associate Physician, Episcopal Hospital. 256 S. Twenty-first St.

‡1907. Council, Malcolm S., M.D., Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Cathcart Home at Devon. Bryn Mawr, Pa.

ELECTED

1924. Cowan, Alfred, M.D., Assistant Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Instructor in Ophthalmology, School of Medicine, University of Pennsylvania; Ophthalmologist, Philadelphia General Hospital. 1930 Chestnut St.

1904. Craig, Frank A., M.D., Instructor in Medicine in the University of Pennsylvania; Visiting Physician to the Henry Phipps Institute, University of Pennsylvania; Visiting Physician to the White Haven Sanatorium; Tuberculosis Consultant to the Presbyterian Hospital. 2011 Pine Street.

1907. Crampton, George S., M.D., Attending Surgeon to the Eye Department of the Pennsylvania Hospital and the Philadelphia Hospital for Contagious Diseases; Assistant Surgeon to the Wills Hospital; Professor of Ophthalmology in the Graduate School of Medicine, University of Pennsylvania; Ophthalmologist to the Philadelphia Orphanage. 2031 Locust St.

1924. Crawford, Baxter Lindsey, M.D., Associate in Pathology, Jefferson Medical College; Chief of Laboratory Service, Jefferson Hospital; Assistant Pathologist, Philadelphia General Hospital. Jefferson Hospital, Tenth and Sansom Sts.

1920. Creighton, William J., B.A., M.D., Assistant Ophthalmic Surgeon, Germantown Hospital and Dispensary; Assistant Dispensary Surgeon, Department Ophthalmology, University Hospital; Instructor in Ophthalmology, University of Pennsylvania Medical School. 258 S. Eighteenth St.

1920. Cross, George Howard, M.D., Ophthalmologist to the Chester Hospital; Associate in Ophthalmology, Graduate School of Medicine, University of Pennsylvania. Chester, Pa.

1917. Cross, Sumner H., A.B., M.D., Physician to the Abington Memorial Hospital, Abington, Pa. Box 126, Jenkintown, Pa.

1921. Crossan, Edward T., M.D., Associate Surgeon, Episcopal Hospital; Associate in Surgery, University of Pennsylvania; Assistant Surgeon Orthopedic Hospital. 257 S. Sixteenth Street.

*1924. Crowell, Bowman C., A.B., M.D., C.M., Associate Director, American College of Surgeons; Director of Clinical Research. 40 E. Erie St., Chicago, Ill.

1904. Cruice, John M., M.D., Physician to St. Agnes and St. Joseph's Hospitals and to the Henry Phipps Institute of the University of Pennsylvania; Instructor in Medicine in the University of Pennsylvania. 406 S. Twenty-second St.

ELECTED

*1910. Cummins, W. Taylor, M.D., Pathologist to the Southern Pacific Hospital; Director of the Mary W. Harriman Research Laboratory, San Francisco, Calif.

1902. Currie, Charles A., M.D., Physician to the Germantown Hospital. West Walnut Lane, Germantown.

1896. Da Costa, John Chalmers, M.D., Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General and St. Joseph's Hospital. 2047 Walnut St.

1887. Daland, Judson, M.D., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 317 S. Eighteenth St.

1919. Davies, John R., Jr., M.D., Chief to the Out-Patient Nose, Throat and Ear Department of the Pennsylvania, Bryn Mawr and Chestnut Hill Hospitals. 302 S. Nineteenth St.

1896. Davis, Charles N., M.D., Dermatologist to the Pennsylvania Hospital; Consulting Dermatologist to St. Agnes' Hospital; Assistant Physician to the Dispensary for Skin Diseases in the Howard Hospital. 1931 Spruce St.

1916. Davis, Warren B., M.D., Rhinologist, Laryngologist, and Otologist, St. Agnes' Hospital; Associate in Surgery, Jefferson Medical College; Assistant Surgeon, Frankford Hospital. 135 S. Eighteenth St.

1900. Davisson, Alex. Heron, M.D., Clinical Chief, City Chest Clinic, No. 12, Rush Hospital. 4514 Springfield Ave.

1894. Deaver, Harry C., M.D., Surgeon to the Episcopal Hospital and to the Children's Hospital of the Mary J. Drexel Home; Surgeon-in-Chief to the Kensington Hospital for Women. 337 S. Eighteenth St.

1887. Deaver, John B., M.D., D.Sc., LL.D., Professor of Surgery in the Graduate School of Medicine; Emeritus Professor of Surgery in the School of Medicine, University of Pennsylvania; Surgeon-in-Chief to the Lankenau Hospital. 1830 DeLancey Street.

1902. Dehoney, Howard, M.D. 240 S. Thirteenth St.

1924. Deichler, L. Waller, M.D., Assistant Surgeon, Wills Hospital; Instructor in Ophthalmology, University of Pennsylvania; Ophthalmologist, Philadelphia General Hospital. 1737 Chestnut St.

1923. De Long, Perce, M.D., Associate in Ophthalmic Pathology in the Graduate School of Medicine, University of Pennsylvania; Assistant Surgeon, Wills Eye Hospital; Assistant Neuro-

ELECTED

1924. Cowan, Alfred, M.D., Assistant Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Instructor in Ophthalmology, School of Medicine, University of Pennsylvania; Ophthalmologist, Philadelphia General Hospital. 1930 Chestnut St.

1904. Craig, Frank A., M.D., Instructor in Medicine in the University of Pennsylvania; Visiting Physician to the Henry Phipps Institute, University of Pennsylvania; Visiting Physician to the White Haven Sanatorium; Tuberculosis Consultant to the Presbyterian Hospital. 2011 Pine Street.

1907. Crampton, George S., M.D., Attending Surgeon to the Eye Department of the Pennsylvania Hospital and the Philadelphia Hospital for Contagious Diseases; Assistant Surgeon to the Wills Hospital; Professor of Ophthalmology in the Graduate School of Medicine, University of Pennsylvania; Ophthalmologist to the Philadelphia Orphanage. 2031 Locust St.

1924. Crawford, Baxter Lindsey, M.D., Associate in Pathology, Jefferson Medical College; Chief of Laboratory Service, Jefferson Hospital; Assistant Pathologist, Philadelphia General Hospital. Jefferson Hospital, Tenth and Sansom Sts.

1920. Creighton, William J., B.A., M.D., Assistant Ophthalmic Surgeon, Germantown Hospital and Dispensary; Assistant Dispensary Surgeon, Department Ophthalmology, University Hospital; Instructor in Ophthalmology, University of Pennsylvania Medical School. 258 S. Eighteenth St.

1920. Cross, George Howard, M.D., Ophthalmologist to the Chester Hospital; Associate in Ophthalmology, Graduate School of Medicine, University of Pennsylvania. Chester, Pa.

1917. Cross, Sumner H., A.B., M.D., Physician to the Abington Memorial Hospital, Abington, Pa. Box 126, Jenkintown, Pa.

1921. Crossan, Edward T., M.D., Associate Surgeon, Episcopal Hospital; Associate in Surgery, University of Pennsylvania; Assistant Surgeon Orthopedic Hospital. 257 S. Sixteenth Street.

*1924. Crowell, Bowman C., A.B., M.D., C.M., Associate Director, American College of Surgeons; Director of Clinical Research. 40 E. Erie St., Chicago, Ill.

1904. Cruice, John M., M.D., Physician to St. Agnes and St. Joseph's Hospitals and to the Henry Phipps Institute of the University of Pennsylvania; Instructor in Medicine in the University of Pennsylvania. 406 S. Twenty-second St.

ELECTED

*1910. Cummins, W. Taylor, M.D., Pathologist to the Southern Pacific Hospital; Director of the Mary W. Harriman Research Laboratory, San Francisco, Calif.

1902. Currie, Charles A., M.D., Physician to the Germantown Hospital. West Walnut Lane, Germantown.

1896. Da Costa, John Chalmers, M.D., Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General and St. Joseph's Hospital. 2047 Walnut St.

1887. Daland, Judson, M.D., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 317 S. Eighteenth St.

1919. Davies, John R., Jr., M.D., Chief to the Out-Patient Nose, Throat and Ear Department of the Pennsylvania, Bryn Mawr and Chestnut Hill Hospitals. 302 S. Nineteenth St.

1896. Davis, Charles N., M.D., Dermatologist to the Pennsylvania Hospital; Consulting Dermatologist to St. Agnes' Hospital; Assistant Physician to the Dispensary for Skin Diseases in the Howard Hospital. 1931 Spruce St.

1916. Davis, Warren B., M.D., Rhinologist, Laryngologist, and Otologist, St. Agnes' Hospital; Associate in Surgery, Jefferson Medical College; Assistant Surgeon, Frankford Hospital. 135 S. Eighteenth St.

1900. Davisson, Alex. Heron, M.D., Clinical Chief, City Chest Clinic, No. 12, Rush Hospital. 4514 Springfield Ave.

1894. Deaver, Harry C., M.D., Surgeon to the Episcopal Hospital and to the Children's Hospital of the Mary J. Drexel Home; Surgeon-in-Chief to the Kensington Hospital for Women. 337 S. Eighteenth St.

1887. Deaver, John B., M.D., D.Sc., LL.D., Professor of Surgery in the Graduate School of Medicine; Emeritus Professor of Surgery in the School of Medicine, University of Pennsylvania; Surgeon-in-Chief to the Lankenau Hospital. 1830 DeLancey Street.

1902. Dehoney, Howard, M.D. 240 S. Thirteenth St.

1924. Deichler, L. Waller, M.D., Assistant Surgeon, Wills Hospital; Instructor in Ophthalmology, University of Pennsylvania; Ophthalmologist, Philadelphia General Hospital. 1737 Chestnut St.

1923. De Long, Perce, M.D., Associate in Ophthalmic Pathology in the Graduate School of Medicine, University of Pennsylvania; Assistant Surgeon, Wills Eye Hospital; Assistant Neuro-

ELECTED

pathologist, Philadelphia General Hospital. 1904 Chestnut Street.

1885. Dercum, Francis X., A.M., M.D., Ph.D., Sc.D., President, American Philosophical Society, Professor Emeritus of Nervous and Mental Diseases in the Jefferson Medical College; Foreign Corresponding Member of the Neurological Society of Paris; Honorary Member of the Psychiatric and Neurological Society of Vienna; Corresponding Member of the Neurological Section of the Royal Society of Medicine; Chevalier de l'Ordre National de la Légion d'Honneur. 1719 Walnut St.

1928. Dewees, A. Lovett, B.A., M.D., Attending Physician, Bryn Mawr Hospital. Haverford, Pa.

1912. Dewey, J. Hiland, Ph.B., M.D., Assistant Surgeon to Wills Hospital; Ophthalmic Surgeon to St. Francis' Hospital, Trenton, N. J. 1436 Diamond St.

*1911. Dickson, Frank D., M.D. 160 Professional Building, Kansas City, Mo.

1908. Dillard, Henry K., Jr., M.D., Physician to the Out-Patient Department of the Pennsylvania Hospital; Physician to the Dispensary of the Mary J. Drexel Home. 234 S. Twentieth Street.

1924. Dillon, Edward S., A.B., M.D., Assistant Physician, Pennsylvania Hospital; Assistant Physician, Department of Metabolism, Philadelphia General Hospital; Instructor in Medicine, School of Medicine, University of Pennsylvania. 2040 Pine St.

1920. Diven, John, A.B., M.D., Pediatrician to St. Christopher's Hospital and Babies' Hospital of Philadelphia. 326 S. Nineteenth St.

1920. Doane, Joseph Chapman, M.D., Associate Professor of Medicine, Temple University; Medical Director, Jewish Hospital; Associate Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 2011 Chestnut St.

*1897. Dorland, W. A. Newman, A.M., M.D., Professor of Gynecology in the Post-graduate Medical School of Chicago; Professor of Obstetrics in the Chicago College of Medicine and Surgery; Visiting Obstetrician to Cook County Hospital. 622 Stratford Place, Chicago, Ill.

1907. Dorrance, George Morris, M.D., Professor of Maxillo-Facial Surgery, Thomas Evans Institute, University of Pennsylvania; Surgeon to St. Agnes' Hospital. 2101 Spruce St.

1902. Downs, Robert N., Jr., M.D., Surgeon to the Dispensary of the Germantown Hospital. 6008 Greene St. Germantown.

ELECTED

1910. Drayton, William, Jr., M.D., Physician to the Philadelphia Hospital for Contagious Diseases; Physician to the Pennsylvania Institute for the Instruction of the Blind. 407 S. Twenty-second Street.

1911. Earnshaw, Henry Culp, M.S., M.D., Attending Physician to the Hospital of the Good Shepherd, Rosemont; Assistant Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Bryn Mawr Children's Hospital; Pennsylvania Railroad Surgeon. Bryn Mawr, Pa.

*1887. Edwards, William A., M.D., Professor of Pediatrics in the Medical Department of the University of California. R. D. 2, Box 105, El Cajon, Calif.

1924. Egbert, Seneca, M.D., Professor of Hygiene, School of Hygiene and Public Health, University of Pennsylvania. Wayne, Pa.

1930. Eiman, John, M.D., Director, Department of Pathology, Presbyterian Hospital; Assistant Professor of Pathology, Graduate School and Associate Department of Pathology, School of Medicine, University of Pennsylvania. Cynwyd, Pa.

1911. Eliason, Eldridge L., B.A., M.D., Sc.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the University Hospital and to the Howard Hospital; Assistant Surgeon to the Philadelphia General Hospital and the American Stomach Hospital. 326 S. Nineteenth St.

1928. Ellison, Richard T., B.S., M.D., Instructor in Medicine, School of Medicine, University of Pennsylvania; Assistant Physician in Charge of Heliotherapy at the Home for Consumptives, Chestnut Hill; Assistant Physician, Presbyterian Hospital. 111 Rex Ave.

1904. Elmer, Walter G., B.S., M.D., Professor of Orthopedics, Graduate School of Medicine, University of Pennsylvania; Clinical Professor of Orthopedic Surgery in the Woman's Medical College of Pennsylvania; Orthopedic Surgeon to the Hospital of the Graduate School of Medicine and to the Philadelphia General Hospital. 1801 Pine St.

1896. Ely, Thomas C., A.M., M.D. 3912 Chestnut St.

†1893. Eshner, Augustus A., M.D., Consulting Physician to Mercy Hospital. 1019 Spruce St.

*1905. Evans, Joseph S., Jr., A.B., M.D., Professor of Medicine, Medical School, University of Wisconsin; Physician to State of Wisconsin General Hospital; Consulting Physician, Madison General and St. Mary's Hospitals. 1300 University Ave., Madison, Wis.

1905. Evans, William, M.D. 500 W. Gay St., West Chester.

ELECTED

1912. Eves, Curtis C., M.D., Otolaryngologist to the Pennsylvania Hospital; Consultant Otolaryngologist to the Philadelphia General Hospital. 1910 Spruce St.

1922. Farley, David L., B.S., M.D., Associate in Medicine, University of Pennsylvania; Physician to Pennsylvania Hospital; Chief of Out-Patient Medical Clinic, Pennsylvania Hospital. 1725 Pine St.

†1903. Farr, Clifford B., A.M., M.D., Director of Laboratories, Pennsylvania Hospital, Department for Mental and Nervous Diseases. Bryn Mawr, Pa.

1893. Farr, William W., M.D., Physician to the Leamy Home. 121 Maplewood Ave., Germantown.

1930. Farrell, John T., Jr., B.S., M.D., Assistant Roentgenologist, Jefferson Hospital; Roentgenologist to Daniel Baugh Institute of Anatomy of Jefferson Medical College. 235 S. Fifteenth St.

1907. Fetterolf, George, A.B., M.D., Sc.D., Professor of Otolaryngology, School of Medicine, University of Pennsylvania. 2010 Spruce St.

1924. Fewell, Alexander G., M.D., Assistant Professor of Ophthalmology, Graduate School of Medicine and Instructor in Ophthalmology in the School of Medicine, University of Pennsylvania; Ophthalmic Surgeon to the Episcopal Hospital. 1924 Pine St.

1907. Fife, Charles A., A.B., M.D., Associate Professor of Pediatrics in the Graduate School of Medicine of the University of Pennsylvania; Pediatrist to the Presbyterian Hospital; Physician to the St. Christopher's Hospital for Children; Assistant Physician to the Philadelphia General Hospital. 2104 Spruce St.

1884. Fisher, Henry M., A.B., M.D. Jenkintown, Pa.

1910. Fisher, John Monroe, M.D., Associate Professor of Gynecology in the Jefferson Medical College; Gynecologist to the Philadelphia, St. Agnes' and Pottstown Hospitals; Assistant Gynecologist to the Jefferson Medical College Hospital. 345 S. Nineteenth St.

1928. Fitz-Hugh, Thomas, Jr., A.B., A.M., M.D., Assistant Attending Physician, University Hospital; Attending Physician, Delaware County Hospital; Instructor in Medicine, University of Pennsylvania. 2020 Locust St.

1928. Flick, John Bernard, M.D., Chief Clinical Assistant in Surgery, Division B, Jefferson Hospital; Assistant Surgeon, Pennsylvania and Bryn Mawr Hospitals; Surgeon, Eagleville

ELECTED

Sanatorium for Consumptives, Eagleville, Pa. 1608 Spruce Street.

1888. Flick, Lawrence F., M.D. 736 Pine St.

1916. Forst, John R., M.D. 166 W. Coulter St., Germantown.

1908. Foulkrod, Collin, M.D., Obstetrician to the Presbyterian Hospital; Assistant Demonstrator of Obstetrics in Jefferson Medical College. 3910 Chestnut St.

1908. Fox, Herbert, M.D., Director of the William Pepper Clinical Laboratory and Professor of Comparative Pathology, School of Medicine, University of Pennsylvania; Pathologist to the Laboratory of Comparative Pathology of the Zoölogical Society of Philadelphia; Pathologist to the Rush Hospital. Haverford, Pa.

†1885. Fox, Joseph M., M.D. Torresdale, Pa.

‡1906. Fraley, Frederick, Jr., A.B., M.D., Associate in Pediatrics, Graduate School of Medicine, University of Pennsylvania. Overbrook, Pa.

1897. Frazier, Charles H., A.B., M.D., Sc.D., John Rhea Barton Professor of Surgery, University of Pennsylvania. 133 S. Thirty-sixth St.

1928. Fuller, Daniel Hunt, A.B., A.M., M.D., Medical Director, Department for Men, Pennsylvania Hospital for Mental and Nervous Diseases. 4401 Market St.

1916. Funk, Elmer Hendricks, M.D., Clinical Professor of Medicine and Therapeutics in the Jefferson Medical College; Assistant Physician to the Pennsylvania and Jefferson Hospitals; Visiting Physician to the White Haven Sanatorium. 269 S. Nineteenth St.

1899. Gamble, Robert, G., M.D., one of the Attending Physicians to the Bryn Mawr Hospital. Haverford, Pa.

1912. Gaskill, Henry Kennedy, M.D. S. E. Cor. Nineteenth and Spruce Sts.

*1913. Gates, Nathaniel, M.D. 10 Peterboro Ave., Detroit, Mich.

1917. Gerhard, Arthur Howell, M.D. 1615 N. Delaware Ave.

1902. Ghriskey, Albert A., M.D. 3936 Walnut St.

1899. Gibbon, John H., M.D., Professor of Surgery in the Jefferson Medical College; Surgeon to the Pennsylvania Hospital and Consulting Surgeon to the Bryn Mawr Hospital. 1608 Spruce Street.

1920. Gill, A. Bruce, A.B., M.D., Professor of Orthopedics, University of Pennsylvania; Surgeon to the Orthopedic Hospital; Orthopedic Surgeon to the Presbyterian Hospital; Chief

ELECTED

Surgeon to the Widener Memorial Industrial Training School for Crippled Children. 1930 Chestnut St.

1927. Gilmour, William Rodger, A.B., A.M., M.D., Assistant Surgeon, Philadelphia General and Methodist Hospitals; Instructor in Anatomy, University of Pennsylvania; Associate Professor of Operative Surgery and Surgical Anatomy, Woman's Medical College. 1737 Chestnut St.

1897. Girvin, John H., M.D., Gynecologist to the Presbyterian Hospital; Associate Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania. 2120 Walnut St.

1920. Githens, Thomas Stotesbury, M.D., Chief Pharmacologist, H. K. Mulford Company. The Cambridge, Wissahickon and School Lane, Germantown.

*1906. Gittings, J. Claxton, M.D., Professor of Pediatrics, School of Medicine and Graduate School of Medicine, University of Pennsylvania; Visiting Physician to the Children's Hospital of Philadelphia. Harwood P.O., Md.

1905. Given, Ellis E. W., M.D., Surgeon to the Philadelphia Freemasons Memorial Hospital of the Masonic Home, Elizabethtown, Pa. 2714 Columbia Ave.

1894. Gleason, E. B., S.B., M.D., LL.D., Professor of Otology in the Graduate School of Medicine, University of Pennsylvania. 2112 Pine St.

1906. Goepp, R. Max, M.D. 518 Medical Arts Building.

1906. von Goldberg, Harold G., M.D., Ophthalmic Surgeon to the Episcopal Hospital and Baltimore & Ohio Railroad; Consulting Ophthalmologist, Misericordia and Kensington Hospitals. 2213 Walnut St.

*1908. Goodman, Edward H., M.D. Dorset, Vt.

1905. Gordon, Alfred, M.D., Neurologist to the Mt. Sinai, the Northwestern General, and the Douglass Memorial Hospitals. 1812 Spruce St.

1928. Gordon, Burgess Lee, A.B., M.D., Director of the Department for Diseases of the Chest, Jefferson Hospital; Assistant Professor in Medicine, Jefferson Medical College. 1832 Spruce St.

1894. Graham, Edwin E., M.D., Professor of Pediatrics in the Jefferson Medical College; Pediatrist to the Jefferson and the Philadelphia General Hospitals; Physician to the Franklin Reformatory Home. 1713 Spruce St.

1926. Grant, Francis C., A.B., M.D., Assistant Professor of Neurosurgery and Associate in Surgery, School of Medicine, University of Pennsylvania; Associate in Neuro-Surgery, Grad-

ELECTED

uate School of Medicine, University of Pennsylvania; Visiting Surgeon, Department of Neurological Surgery, Philadelphia General Hospital. 133 S. Thirty-sixth St.

1904. Grayson, Charles P., M.D., Emeritus Professor of Laryngology in the University of Pennsylvania; Otolaryngologist to the Philadelphia General Hospital. 262 S. Fifteenth St.

1910. Greenman, Milton J., M.D., Sc.D., Director of the Wistar Institute of Anatomy and Biology. Wistar Institute of Anatomy and Biology, Thirty-sixth St. and Woodland Ave.

1883. Griffith, J. P. Crozer, A.B., M.D., Ph.D., Professor of Pediatrics in the Graduate School of Medicine, University of Pennsylvania; Corresponding Member of the Société de Pédiatrie de Paris; Physician to the Children's Hospital of Philadelphia. 1810 Spruce St.

1912. Griscom, J. Milton, B.S., M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Attending Surgeon to the Wills Hospital. 2213 Walnut St.

1911. Gummey, Frank Bird, M.D., Visiting Physician to the Germantown Hospital and Dispensary; Visiting Physician to the Midnight Mission. 5418 Greene St., Germantown.

*1902. Gwyn, Norman B., M.D., Clinician in Medicine, Toronto General Hospital; Clinician in Medicine, Medical Faculty, University of Toronto School. 109 Madison Ave., Toronto, Canada.

1894. Hamill, Samuel McC., M.D., Pediatrist to the Presbyterian Hospital; Pediatrist to St. Vincent's Home. 1822 Spruce St.

1897. Hand, Alfred, Jr., M.D., Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania; Consulting Physician to the Children's Hospital; Visiting Physician to the Children's Hospital of the Mary J. Drexel Home; Pediatrist to the Widener Memorial School; Consulting Pediatrist to the Methodist Hospital. 1724 Pine St.

1886. Hansell, Howard F., M.D., Emeritus Professor of Ophthalmology in the Jefferson Medical College. N. E. Cor. Seventeenth and Walnut Sts.

1889. Hare, Hobart A., M.D., LL.D., Professor of Therapeutics, Materia Medica, and Diagnosis in the Jefferson Medical College. 1801 Spruce St.

1929. Harrison, Francis G., M.D., Assistant Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Assistant Urologist, Out-Patient Department, Presbyterian Hospital. 1900 Spruce St.

ELECTED

1903. Hart, Charles D., A.M., M.D., Inspector and Secretary of the Eastern State Penitentiary; National Executive Committee and Chairman of the Philadelphia Committee, Boy Scouts of America. Chestnut Hill.

1926. Hartman, Frederick L., A.B., M.D., Clinical Associate in Medicine, Lankenau Hospital; Assistant Instructor in Pathology, University of Pennsylvania; Assistant Pathologist, Visiting Staff of University and Philadelphia General Hospitals. 1914 Pine St.

1907. Hatfield, Charles James, A.B. (Princeton), M.D., Executive Director of the Henry Phipps Institute for the Study, Treatment, and Prevention of Tuberculosis; Visiting Physician to the White Haven Sanatorium. Phipps Institute, Seventh and Lombard Sts.

*1928. Hayman, Joseph M., Jr., A.B., M.D., Associate Professor of Medicine, Western Reserve University; Associate Physician, Lakeside Hospital. Cleveland, Ohio.

1911. Heed, Charles R., M.D., Ophthalmologist to Girard College; Assistant Ophthalmologist to the Jefferson Medical College Hospital. 1205 Spruce St.

1901. Heisler, John C., M.D., Professor of Anatomy, University of Pennsylvania. 3829 Walnut St.

1903. Henry, J. Norman, M.D., Physician to the Pennsylvania Hospital; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania; Assistant Physician to the Philadelphia General Hospital. 1906 Spruce St.

1927. Herman, Leon, B.S., M.D., Urologist, Methodist Hospital; Assistant Surgeon in Charge of Urology, Pennsylvania Hospital. 801 Medical Arts Building.

1891. Hewson, Addinell, A.B., A.M., M.D., Professor of Anatomy and Histology in the Temple University; Surgeon to Memorial Hospital, Roxborough. 257 S. Sixteenth St.

1924. Hewson, William, A.B., M.D., Assistant in Otology, Graduate School of Medicine, University of Pennsylvania; Otolaryngologist, Pennsylvania Institution for Instruction of Blind and the Shriners' Hospital for Crippled Children; Assistant Otolaryngologist, Memorial Hospital. 6013 Greene St., Germantown.

1909. Higbee, William S., M.D., President of the Pennsylvania State Board of Examiners for Registration of Nurses. 5121 Pine St.

1897. Hinkle, William M., M.D., Associate in Otology in the Graduate School of Medicine, University of Pennsylvania; Lecturer on the Anatomy and Physiology of the Vocal Organs in the

ELECTED

National School of Elocution and Oratory. 923 N. Sixty-fourth St.

*1892. Hinsdale, Guy, A.M., M.D. White Sulphur Springs, W. Va.

1888. Hirst, Barton Cooke, A.B., M.D., LL.D., Professor of Obstetrics, Graduate School of Medicine, University of Pennsylvania; Gynecologist to the Philadelphia General and the Howard Hospitals. 1821 Spruce St.

1924. Hirst, John C., 2nd, A.B., M.D., Assistant Professor of Obstetrics, School of Medicine and Graduate School of Medicine, University of Pennsylvania; Assistant Obstetrician, University Hospital; Obstetrician to St. Agnes' Hospital; Gynecologist to Orthopedic Hospital. 1918 Pine St.

*1908. Hitchens, Arthur Parker, M.D., M.C., U.S.A., in charge, 6th Corps Area. Laboratory, Fort Sheridan, Ill.

1905. Hodge, Edward Blanchard, A.B., M.D., Surgeon to the Presbyterian and the Germantown Hospitals; Associate Professor of Surgery in the Graduate School of Medicine, University of Pennsylvania; Associate Surgeon to the Pennsylvania Hospital; Surgeon-in-Chief to Chester County Hospital, West Chester, Pa. 2019 Spruce St.

1906. Holloway, Thomas B., M.S., M.D., Sc.D., Professor of Ophthalmology, School of Medicine, University of Pennsylvania; Ophthalmologist, Orthoepdic Hospital, and in Department of Neurology, Philadelphia General Hospital; Ophthalmologist to the Pennsylvania Institution for the Instruction of the Blind at Overbrook. 1819 Chestnut Street.

1914. Hooker, Richard S., M.D., Assistant Physician to the Episcopal Hospital, and to the Philadelphia Orthopedic Hospital and Infirmary for Nervous Diseases. 2147 N. Howard St.

1919. Hopkins, Arthur H., B.S. (Haverford), M.D. 1726 Pine St.

1925. Hopkinson, R. Dale, M.D., Registrar and Assistant Physician, Abington Memorial Hospital. Jenkintown, Pa.

1928. Houser, Karl M., Ph.B., M.D., M.Sc., Instructor in Otolaryngology, University of Pennsylvania; Assistant in Otolaryngology, University Hospital; Otolaryngologist, Delaware County Hospital. 1826 Pine St.

1908. Hoyt, Daniel M., M.D., Assistant Visiting Physician to the Philadelphia General Hospital. The Tracy, Thirty-sixth and Chestnut Sts.

*1912. Huber, G. Carl, M.D., Professor of Anatomy and Director of the Anatomic Laboratories and Dean of the Graduate School, University of Michigan. 1330 Hill St., Ann Arbor, Mich.

ELECTED

1892. Hughes, William E., M.D., Visiting Physician to the Philadelphia General Hospital; Consulting Physician to the Presbyterian Hospital. 3945 Chestnut St.

1923. Hume, John E., A.B., M.D., Visiting Physician, Medical Department, Howard Hospital. N. E. Cor. Twentieth and Chestnut Sts.

*1912. Hunter, John W., B.S., M.D. McDaniel, Md.

1921. Hunter, Robert J., M.D., Laryngologist, Philadelphia General Hospital; Associate Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania. 2011 Chestnut Street.

1926. Husik, David N., M.D., Professor of Otology, Graduate School of Medicine, University of Pennsylvania; Laryngologist, Philadelphia General, Mount Sinai and American Stomach Hospitals. 1930 Chestnut St.

1898. Hutchinson, James P., M.D., Consulting Surgeon to the Pennsylvania and to the Methodist Hospitals. Media, Pa.

1921. Ivy, Robert H., D.D.S., M.D., Professor of Maxillo-Facial Surgery, Graduate School of Medicine, University of Pennsylvania; Plastic Surgeon to the Hospitals of the Graduate School of Medicine, University of Pennsylvania; Oral Surgeon, Episcopal Hospital; Consultant in Maxillo-Facial Surgery, Walter Reed General Hospital, Washington, D.C. 1930 Chestnut St.

1917. Jackson, Chevalier, M.D., Sc.D., LL.D., Professor of Bronchoscopy and Esophagoscopy, Temple University School of Medicine; Bronchoscopist and Esophagoscopist, Temple University Hospital; William Potter Memorial Lecturer, Jefferson Medical College; Lecturer on Bronchoscopy and Esophagoscopy, Woman's Medical College. 235 S. Fifteenth St.

*1885. Jackson, Edward, A.M., M.D., Sc.D., Emeritus Professor of Ophthalmology in the University of Colorado; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic; Secretary of State Commission for the Blind of Colorado. 217 Imperial Building, Denver, Colo.

1906. Jacobs, Francis Brinton, B.S., M.D., Pediatrist to the Chester County Hospital. West Chester, Pa.

1922. Jameson, Howard Leon, M.D., Associate Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 2133 S. Sixteenth St.

ELECTED

1920. Janvier, G. Victor, M.D., Associate in Obstetrics, School of Medicine, University of Pennsylvania; Assistant Obstetrician, University Hospital; Assistant Obstetrician and Gynecologist, Philadelphia General and Howard Hospitals; Cystoscopist to the Obstetric and Gynecologic Department of the Delaware County Hospital. Lansdowne, Pa.

1913. Jefferys, William Hamilton, A.B., A.M., M.D. Rosemont, Pa.

1919. Jenks, Horace H., A.B., M.D., Medical Director, Associated Medical Clinics; Assistant Visiting Physician to the Children's Hospital; Assistant Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania; Physician to St. Christopher's Hospital for Children. 918 Clinton St.

1930. Jepson, Paul N., A.B., M.D., Instructor in Orthopedic Surgery, School of Medicine, University of Pennsylvania; Assistant Orthopedic Surgeon, University Hospital; Associate Orthopedic Surgeon, Jewish Hospital and Graduate Hospital of the University of Pennsylvania. 1824 Spruce St.

1928. John, Rutherford Lewis, B.S., M.D., Instructor in Surgery, School of Medicine, University of Pennsylvania; Associate Surgeon in Charge of Orthopedics, Episcopal Hospital; Orthopedic Surgeon to St. Christopher's Hospital; Assistant Surgeon to the Orthopedic Hospital and Infirmary for Nervous Diseases. 256 S. Twenty-first St.

1926. Johnson, George Ernest, M.D. 5341 Chester Ave.

1898. Johnson, Russell H., A.B. (Princeton), M.D., Physician to the Pennsylvania Institution for the Deaf and Dumb. Chestnut Hill, Philadelphia.

1928. Johnson, William N., A.B., M.D., Consulting Physician, Germantown Hospital. 6430 Germantown Ave.

1918. Jonas, Leon, M.D. 1726 Pine St.

1900. Jones, Charles James, A.M., M.D., LL.D., Ophthalmic Surgeon to St. Joseph's Hospital; Ophthalmic Surgeon to the House of the Good Shepherd, Germantown; Consulting Ophthalmologist to St. Vincent's Home. 626 Medical Arts Building.

1924. Jones, Harold W., M.D., Demonstrator of Medicine, Jefferson Medical College; Assistant Physician, Philadelphia General Hospital. 1930 Chestnut St.

1913. Jones, John F. X., B.S., A.B., A.M., M.D., Surgeon to St. Joseph's, Misericordia and St. Agnes' Hospitals. 1737 Chestnut St.

ELECTED

1900. Jopson, John H., M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Presbyterian Hospital; Consulting Surgeon to Children's and Bryn Mawr Hospitals. 1824 Pine St.

1900. Judson, Charles F., A.B., M.D., Physician to Out-Patient Department of the Lankenau Hospital and to the Southern Home for Children. 1005 Spruce St.

1902. Jump, Henry D., M.D., Professor of Applied Therapeutics and Clinical Medicine, Woman's Medical College; Physician to Philadelphia General and Misericordia Hospitals. 2019 Walnut St.

1903. Kalteyer, Frederick J., M.D., Associate Professor of Medicine, Jefferson Medical College; Assistant Physician, Jefferson Medical College Hospital; Consulting Physician to the Pottstown Hospital, Pottstown, Pa. 1530 Locust St.

*1910. Karsner, Howard T., M.D., Professor of Pathology and Director of Pathology of the Institute of Pathology, Western Reserve University. 2109 Adelbert Rd., Cleveland, Ohio.

1926. Kaufman, Isadore, M.D., Associate in Medicine, University of Pennsylvania; Medical Director, Kensington Dispensary for Tuberculosis; Associate Clinical and Sociological Director, Phipps Institute; Visiting Physician to White Haven Sanatorium. 1616 Walnut St.

1923. Kay, James, M.D., Visiting Physician, Episcopal Hospital; Associate Professor in Physical Diagnosis, Medical School, Temple University. 600 W. Olney Ave.

1924. Keeler, J. Clarence, M.D., Associate Professor of Otology, Jefferson Medical College and Hospital; Assistant in Otolaryngology, Germantown Hospital; Consultant Otolaryngologist, Pottstown Hospital, Pa., and Newcombe Hospital, Vineland, N. J. Medical Arts Building.

†1867. Keen, William W., M.D., LL.D. (Hon.), Ph.D. (Brown, Upsala, etc.), (Hon.) Sc.D. (Jefferson and Harvard), Dr., Honoris Causa, University of Paris; (Hon.) F.R.C.S. (Eng., Edin. and Ireland). 1520 Spruce St.

1912. Keene, Floyd E., M.D., Goodell Professor of Gynecology in the School of Medicine, and Professor of Gynecology in the Graduate School of Medicine, University of Pennsylvania; Gynecologist to the University Hospital; Gynecologist to the Chestnut Hill Hospital. 133 S. Thirty-sixth St.

1913. Kelly, Francis Joseph, M.D. 2211 Locust St.

ELECTED

*1887. Kelly, Howard A., A.B., M.D., LL.D. (Aberdeen, Wash. and Lee, and Univ. of Pa.), Professor of Gynecology in Johns Hopkins University and Gynecologist to the Johns Hopkins Hospital, Baltimore, Md.; Hon. Fellow of the Edinburgh Obstetrical Society, the Royal Academy of Medicine of Ireland, and of the Glasgow Obstetrical and Gynecological Society. 1418 Eutaw Place, Baltimore, Md.

1909. Kelly, James A., A.M., M.D.,Associate Professor of Surgery in the Graduate School of Medicine, University of Pennsylvania; Visiting Surgeon to St. Mary's, St. Joseph's and Misericordia Hospitals. 1900 S. Rittenhouse Square.

1912. Kelly, Thomas C., A.M., M.D., Associate in Medicine, University of Pennsylvania; Assistant Professor of Medicine, Graduate School, University of Pennsylvania; Pediatrist to St. Christopher, Misericordia and St. Mary's Hospitals; Assistant Physician, Philadelphia General Hospital. 105 School Lane, Germantown.

1898. Kempton, Augustus F., M.D. 2118 Pine St.

1905. Kercher, Delno E., M.D. 1534 Pine St.

1921. Kern, Richard A., A.B., M.D., Assistant Professor of Medicine, University of Pennsylvania; Assistant Physician, University Hospital; Associate in Medicine, Graduate School of Medicine, University of Pennsylvania. University Hospital, Thirty-sixth and Spruce Sts.

1930. Kimbrough, Robert A., Jr., A.B., M.D., Associate in Obstetrics and Gynecology, University of Pennsylvania; Assistant Gynecologist, University Hospital; Assistant Obstetrician, Pennsylvania Hospital; Consultant Obstetrician, Chester County Hospital. 1907 Spruce St.

1920. Klauder, Joseph Victor, A.B., M.D., Professor of Dermatology, Woman's Medical College; Associate Professor, Dermatology and Syphilology, Graduate School of Medicine, University of Pennsylvania; Dermatologist, Misericordia, Wills and Abington Memorial Hospitals; Chief of Clinic, Philadelphia General Hospital. 1934 Spruce St.

1920. Klein, Thomas, M.D., Professor of Applied Therapeutics, Medical School, Temple University; Associate Professor of Medicine, Graduate School, University of Pennsylvania; Physician to Samaritan and Philadelphia General Hospitals. 250 S. Eighteenth St.

1913. Klopp, Edward J., M.D., Associate in Surgery in the Jefferson Medical College; Assistant Surgeon to the Jefferson Hospital; Surgeon and Chief of the Out-Patient Clinic of the Pennsyl-

ELECTED

vania Hospital; Surgeon to the Delaware County Hospital. 1611 Spruce St.

1908. Knipe, Jay C., M.D., Ophthalmologist to the Jewish and Mary J. Drexel Hospitals; Associate in Ophthalmology, Jefferson Medical College; Chief of Eye Clinic, Jefferson Hospital. 1737 Chestnut St.

1919. Knipe, Norman Leslie, Ph.B. (Cornell); M.D., Gynecologist, St. Mary's Hospital; Associate Gynecologist, American Stomach Hospital. 2018 Chestnut St.

1908. Knowles, Frank Crozer, M.D., Professor of Dermatology, Jefferson Medical College; Dermatologist to the Presbyterian and Children's Hospitals; Chief of Clinic, Pennsylvania Hospital. 2035 Spruce St.

1925. Knox, Harry E., M.D., Surgeon to St. Christopher's Hospital; Surgeon to Philadelphia Hospital for Contagious Diseases; Associate Surgeon, Episcopal Hospital; Assistant Surgeon, Germantown Hospital. 719 Sixty-sixth Ave., Oak Lane, Phila.

1914. Kolmer, John A., M.D., Dr. P.H., M.Sc., D.Sc., LL.D., Professor of Pathology and Bacteriology, Graduate School of Medicine, University of Pennsylvania; Head of the Department of Pathology and Bacteriology Research Institute for Cutaneous Medicine; Consulting Pathologist to St. Vincent's, Misericordia, Memorial and Jeanes Hospitals. Cynwyd, Pa.

1904. Krauss, Frederick, M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmic Surgeon to St. Christopher's Hospital for Children; Laryngologist to the Abington Hospital. 1930 Chestnut St.

1914. Krumbhaar, Edward B., A.B., Ph.D., M.D., Professor of Pathology, School of Medicine, and Graduate School of Medicine, University of Pennsylvania. Box 4310, Chestnut Hill.

1900. Krusen, Wilmer, M.D., Professor of Gynecology in the Medical Department of Temple University; Chief Gynecologist to the Samaritan and the Garretson Hospitals; Consulting Gynecologist to the Charity and Mercy Hospitals. Lansdowne, Pa.

1904. Landis, Henry R. M., M.D., Professor of Clinical Medicine in the School of Medicine, and Director of the Clinical and Sociological Departments of the Henry Phipps Institute of the University of Pennsylvania; Visiting Physician to the White Haven Sanatorium. 1817 Pine St.

1907. Langdon, H. Maxwell, M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania

ELECTED

Instructor of Ophthalmology, School of Medicine, University of Pennsylvania; Ophthalmologist of the Presbyterian, Orthopedic and Children's Hospitals. 1530 Locust St.

1922. Lawrance, Stuart J., M.D., Obstetrician, St. Mary's Hospital. 1332 Spruce St.

1926. Laws, George M., B.S., M.D., Associate Gynecologist, Presbyterian Hospital; Gynecologist, American Hospital for Diseases of the Stomach. 1907 Spruce St.

1920. Leavitt, Frederick Headley, M.D., Associate in Neurology, Graduate School of Medicine, University of Pennsylvania; Assistant Physician to the Orthopedic Hospital and Infirmary for Nervous Diseases. 1527 Pine St.

1904. Le Boutillier, Theodore, M.D., Assistant Professor, Epidemic Contagious Diseases, Graduate School of Medicine, University of Pennsylvania; Senior Pediatrist, Philadelphia Hospital for Contagious Diseases; Visiting Physician, Babies' Hospital, North Philadelphia. 1930 Chestnut St.

1908. Lee, Walter Estell, M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Pennsylvania, Germantown, Bryn Mawr and Children's Hospitals, and to the Hospital of the Graduate School of Medicine, University of Pennsylvania. 905 Pine St.

‡1903. Leffmann, Henry, A.M., M.D., D.D.S., Ph.D., Honorary Professor of Chemistry, Wagner Free Institute of Science; Emeritus Pathological Chemist, Jefferson Hospital; Lecturer on Research, Philadelphia College of Pharmacy and Science. 1729 N. Seventeenth St.

1892. Leidy, Joseph, M.D., Officier d'instruction publique, France; Consulting Physician to the Pennsylvania Training School for Feeble-minded Children. 1713 Walnut St.

1920. Leopold, Simon S., M.D., Associate in Medicine, University of Pennsylvania; Assistant Physician, University Hospital. 2025 Spruce St.

1915. Lewis, Fielding O., M.D., Associate in Laryngology in the Jefferson Medical College Hospital; Operating Clinical Chief of the Laryngological Dispensary and Clinical Assistant of the Otological Dispensary of the Jefferson Medical College Hospital; Laryngologist of the Philadelphia General Hospital. 259 S. Seventeenth St.

1904. Lindauer, Eugene, M.D., Assistant Neurologist to the Philadelphia General and Polyclinic Hospitals; Instructor in Neurology, Graduate School of Medicine, University of Pennsylvania. 6451 N. Broad St.

ELECTED

1927. Lindsey, Walter H., A.B., M.D., Associate Physician, Episcopal Hospital; Assistant Medical Director, Philadelphia Rapid Transit Company. S. E. Cor. Seventeenth and Arch Streets.

1886. Lloyd, J. Hendrie, A.M. (Princeton), M.D., Consulting Neurologist to the Methodist Episcopal Hospital; Consulting Neurologist to the State Asylum for the Chronic Insane at Wernersville, and to the Pennsylvania Training School for Feeble-minded Children at Elwyn. 4057 Spruce St.

1907. Lodholz, Edward, M.D., Isaac Ott Professor of Physiology, Graduate School of Medicine, University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1893. Longaker, Daniel, M.D., Consulting Obstetrician to the Kensington Hospital for Women. 1806 Spruce St.

‡1907. Loux, Hiram R., M.D., Professor of Genito-urinary Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General Hospital. Medical Arts Building, Sixteenth and Walnut Sts.

1921. Lucké, Baldwin, Dr. P.H., M.D., Assistant Pathologist, University and Philadelphia General Hospitals; Associate Professor of Pathology, School of Medicine, and Assistant Professor of Pathology in the Graduate School of Medicine, University of Pennsylvania. Medical School, University of Pennsylvania.

1924. Ludy, John B., A.B., A.M., M.D., Dermatologist to Pennsylvania, Lankenau, Methodist and Delaware County Hospitals. Medical Arts Building.

1923. Lueders, Charles W., A.B., M.D., Gastroenterologist and Chief of Clinic, Pennsylvania Hospital. 1930 Chestnut St.

1930. Lukens, Robert M., M.D. 1308 Hunting Park Ave.

1929. Lull, Clifford B., M.D., Demonstrator in Obstetrics, Jefferson Medical College; Attending Physician, Department of Gynecology and Obstetrics, Philadelphia General and Delaware County Hospitals; Assistant Obstetrician, Lying-in and Jefferson Hospitals. 1731 Pine St.

1919. Lynch, Frank B., Jr., M.D., Dickson Fellow in Bacteriology to the Pepper Laboratory of Clinical Medicine, University of Pennsylvania; Serologist, University Hospital; Instructor in Pathology, Evans Institute, University of Pennsylvania. Pepper Laboratory, University of Pennsylvania.

1914. Lyon, B. B. Vincent, A.B. (Williams Coll.), M.D. (Johns Hopkins), Associate in Medicine, Jefferson Medical College; Chief-of-Clinic, Gastro-Intestinal Department, Jefferson Hospital. 2031 Locust St.

ELECTED

1900. McCarthy, Daniel J., M.D., Professor of Medical Jurisprudence (George B. Wood Foundation) in the University of Pennsylvania; Neurologist to the Philadelphia General and St. Agnes' Hospitals, and to the Henry Phipps Institute. 2025 Walnut St.

1913. McCrae, Thomas, B.A., M.D., F.R.C.P. (Lond.), Professor of Medicine in the Jefferson Medical College; Physician to the Jefferson and Pennsylvania Hospitals. 1929 Spruce Street.

1895. McFarland, Joseph, M.D., Sc.D., Professor of Pathology in the School of Medicine, University of Pennsylvania; Pathologist to the Philadelphia General Hospital. 542 W. Hortter St., Germantown.

1905. McKenzie, R. Tait, M.D., LL.D., Professor of Physical Education and Director of the Department of Physical Education in the University of Pennsylvania. 2014 Pine St.

*1916. McKnight, Howard A., A.B., M.D. Tecumseh Building, Springfield, Ohio.

1915. McLean, John D., M.D. 135 S. Thirty-sixth St.

1924. McMillan, Thomas M., A.B., M.D., Assistant Physician, Pennsylvania Hospital; Cardiologist, Philadelphia General Hospital; Instructor in Medicine, School of Medicine, University of Pennsylvania; Associate Professor of Cardiology, Graduate School of Medicine, University of Pennsylvania. 2044 Locust St.

1924. McPhedran, F. Maurice, A.B., M.D., Associate in Medicine, School of Medicine, University of Pennsylvania; Assistant Professor of Medicine, Henry Phipps Institute. Seventh and Lombard Sts.

*1900. McReynolds, Robert Phillips, M.D. 213 S. Broadway, Los Angeles, Calif.

1910. Mackinney, William H., M.D., Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Genito-urinary Surgeon, Philadelphia General Hospital; Chief of Urologic Clinic, Lankenau Hospital. 1930 Chestnut St.

1901. MacLeod, George I., M.D. 131 S. Twenty-second St.

1914. Maier, F. Hurst, M.D., Associate in Gynecology to the Jefferson Medical College; Gynecologist to St. Joseph's Hospital; Gynecologist, Obstetrician and Consultant, Prenatal Clinic, Philadelphia General Hospital; Associate Gynecologist, St. Mary's Hospital. 2019 Walnut St.

1913. Major, Charles P., B.S., M.D., Pediatrist to the Medical Service of the Germantown Hospital; Pediatrist to the Abington Memorial Hospital. Tenth and Oak Lane.

ELECTED

1913. Manges, Willis F., D.Sc., M.D., Roentgenologist to the Jefferson Hospital; Clinical Professor of Roentgenology, Jefferson Medical College. 235 S. Fifteenth St.

1898. Marshall, George Morley, M.D., Laryngologist to the Philadelphia General Hospital; Laryngologist and Otologist to St. Joseph's Hospital; Associate Professor of Laryngology in the Graduate School of Medicine, University of Pennsylvania. 1819 Spruce St.

1889. Martin, Edward, M.D., Emeritus Professor of Surgical Physiology, University of Pennsylvania. 135 S. Eighteenth St.

*1911. Meigs, Edward Browning, A.B., M.D., Physiologist in the Dairy Division of the United States Department of Agriculture. 1736 M. St., N. W., Washington, D. C.

1914. Mencke, J. Bernhard, A.B., M.D., Assistant Surgeon to the Lankenau Hospital; Surgeon to the Out-Patient Department of the Lankenau Hospital. 1816 Spruce St.

1923. Mendelson, Walter, M.D. 639 Church Lane, Germantown.

1914. Merrill, William Jackson, A.B., M.D., Associate Professor of Orthopedics, Graduate School of Medicine, University of Pennsylvania; Orthopedic Surgeon to the Children's Hospital, Jewish Hospital, Children's Seashore House, Lock Haven Hospital, City Hospital, Williamsport, Pa., and Geisinger Memorial Hospital, Danville, Pa. 2017 Spruce St.

*1894. Miller, D. J. Milton, M.D., Associate Physician to the Children's Hospital, Philadelphia; Pediatrist to the Bamberger Home for Invalid Children, Longport, N. J. N. W. Cor. Pacific and California Aves., Atlantic City, N. J.

1922. Miller, Edwin B., M.D., Ophthalmologist to Stetson and Northwestern General Hospitals. 2008 Walnut St.

1921. Miller, T. Grier, A.B., M.D., Assistant Professor of Medicine, University of Pennsylvania; Chief, Gastro-intestinal Clinic; Assistant Physician, University Hospital; Consultant, Friends' Hospital and Shriners' Hospitals for Crippled Children. 318 University Hospital.

1881. Mills, Charles K., M.D., LL.D., Emeritus Professor of Neurology in University of Pennsylvania; Neurologist to Philadelphia General Hospital; Consulting Physician to the Orthopedic Hospital and Infirmary for Nervous Diseases. 2121 DeLancey St.

*1917. Mitchell, A. Graeme, M.D., Professor of Pediatrics, College of Medicine, University of Cincinnati; Director of Pediatric and Contagious Services, Cincinnati General Hospital; Chief of Staff of Children's Hospital, Cincinnati. University of Cincinnati.

ELECTED

1904. Mitchell. Charles F., M.D., Surgeon to the Pennsylvania, Bryn Mawr, and Germantown Hospitals; Associate Professor of Surgery in the Graduate School of Medicine, University of Pennsylvania. 2003 Pine St.

1921. Mohler, Henry K., P.D., M.D., Associate in Medicine, Jefferson Medical College; Medical Director, Jefferson Hospital. 319 S. Sixteenth St.

*1908. Montgomery, Charles M., A.B., M.D. Oteen, N. C.

1928. Moon, Virgil Holland, A.B., M.D., M.Sc. Jefferson Medical College.

1923. Moore, William F., M.D., Assistant, Bronchoscopic Clinic, Jefferson Hospital; Bronchoscopist to Philadelphia General Hospital and Philadelphia Hospital for Contagious Diseases; Assistant Laryngologist, Children's Hospital, Out-Patient Department; Laryngologist to Tuberculosis Department, Philadelphia General Hospital. 255 S. Sixteenth St.

1924. Moorhead, Stirling W., A.B., M.D., Associate in Urology, School of Medicine, University of Pennsylvania; Assistant Surgeon, Department of Genito-urinary Surgery and in Urological Dispensary, University Hospital; Urologist, Howard Hospital. 1523 Pine St.

1918. Morgan, Arthur C., M.D., Former Professor of Clinical Medicine, Temple University; Visiting Physician to Tuberculosis Department, Philadelphia General Hospital; Consulting Physician to the Eastern State Penitentiary and to the Frankford Hospital. 1930 Chestnut St.

1886. Morris, Caspar, M.D. Haverford, Pa.

1893. Morris, Elliston J., M.D., Consulting Physician to the Episcopal Hospital. 128 S. Eighteenth St.

‡1897. Morton, Samuel W., M.D. 2049 Chestnut St.

1929. Mudd, Stuart, B.S., M.A., M.D., Associate in Pathology, Henry Phipps Institute; Assistant Professor of Experimental Pathology, University of Pennsylvania. Villa Nova, Pa.

1905. Müller, George P., M.D., Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the University and Misericordia Hospitals. 1930 Spruce St.

1930. Murphy, Douglas P., M.D., Fellow in Research, Gynecean Hospital Institute of Gynecological Research, University of Pennsylvania. 4422 Osage Ave.

*1915. Musser, John H., Jr., B.S., M.D., Professor of Medicine and Head of Department, School of Medicine, Tulane University of Louisiana. 1430 Tulane Ave., New Orleans, La.

ELECTED

1905. Mutschler, Louis H., M.D., Surgeon to the Episcopal Hospital; Associate Surgeon to the Orthopædic Hospital. 1625 Spruce Street.

1902. Nassau, Charles F., M.D., LL.D., Associate Professor of Surgery in the Jefferson Medical College; Surgeon to St. Joseph's Hospital; Chief Surgeon to the Frankford Hospital; Surgeon to Girard College. 1710 Locust St.

‡1886. Neff, Joseph S., A.M., M.D., LL.D., D.P.H., Narberth, Pa.

1887. Neilson, Thomas Rundle, A.M., M.D., Surgeon Emeritus to the Episcopal Hospital; Consulting Surgeon to St. Christopher's Hospital for Children; Emeritus Professor of Genito-urinary Surgery in the University of Pennsylvania. 1937 Chestnut St.

1905. Newcomet, William S., M.D., Physician in Charge of Radiological Department, Jefferson, Presbyterian and Oncologic Hospitals. 3501 Baring St.

1905. Newlin, Arthur, B.S., M.D., Physician to the Pennsylvania Hospital. 1804 Pine St.

1899. Nicholson, William Rufus, A.B., M.D., Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania; Attending Gynecologist and Attending Obstetrician to the Methodist Episcopal Hospital; Attending Obstetrician to the Presbyterian Hospital. 2023 Spruce St.

1889. Noble, Charles P., M.D., Sc.D. (Hon.), Formerly Surgeon-in-Chief, Kensington Hospital for Women; Formerly Clinical Professor of Gynecology, Woman's Medical College. Radnor, Pa.

1905. Norris, Charles C., M.D., Director of the Department and Professor of Obstetrics and Gynecology in the School of Medicine, and Professor of Gynecology in the Graduate School of Medicine, University of Pennsylvania; Gynecologist and Obstetrician to the University Hospital; Director, Gynecean Hospital Institute of Gynecological Research, University of Pennsylvania; Gynecologist to Henry Phipps Institute and to the Children's Hospital. 133 S. Thirty-sixth St.

1905. Norris, George William, A.B., M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician-in-Chief, Medical Service "A", Pennsylvania Hospital; Attending Specialist (Heart and Lungs), U. S. Public Health Service (Veterans' Bureau). 321 S. Eleventh St.

*1901. Norris, Henry, M.D., Surgeon to the Rutherford Hospital. Waverly Mills, S. C.

ELECTED

1892. Norris, Richard C., M.D., Professor of Obstetrics in the Graduate School of Medicine, University of Pennsylvania; Obstetrician in Charge of the Preston Retreat; Visiting Obstetrician to the Philadelphia General Hospital; Gynecologist to the Methodist Episcopal Hospital, and Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital. 500 N. Twentieth St.

1913. O'Neal, Alexander H., A.B., A.M., M.D., Physician to the Cathcart and Richardson Homes, Devon; Anesthetizer to the Bryn Mawr Hospital. St. Davids, Pa.

1924. Opie, Eugene L., A.B., M.D., Professor of Pathology and Director of the Department of Pathology in the School of Medicine, University of Pennsylvania; Director of Laboratories, Henry Phipps Institute. Phipps Institute, Seventh and Lombard Streets.

1903. Ostheimer, Maurice, A.B., M.D., Whitford, Pa.

1913. Outerbridge, George W., A.B., M.D., Associate Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania; Assistant Gynecologist to the Methodist Hospital; Gynecologist to the Abington Memorial Hospital. 1927 Spruce St.

1915. Owen, Hubley R., M.D., Professor of Clinical Surgery, Woman's Medical College; Surgeon, Philadelphia General Hospital; Assistant Surgeon, Orthopedic Hospital; Chief Surgeon, Bureaus of Police and Fire, Philadelphia. 319 S. Sixteenth St.

1897. Packard, Francis R., M.D., Chief of the Department of Diseases of the Nose and Throat, Pennsylvania Hospital; Consulting Otolaryngologist to the Children's Hospital of Philadelphia; Consulting Laryngologist, Bryn Mawr Hospital; Editor, *Annals of Medical History*. 304 S. Nineteenth St.

1898. Page, Henry F., M.D., Assistant Physician to the Lankenau Hospital and Physician to the Medical Dispensary of the same; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania. 315 S. Sixteenth St.

1906. Pancoast, Henry K., M.D., Professor of Roentgenology in the School of Medicine, University of Pennsylvania and Roentgenologist to the University Hospital. Haverford, Pa.

1909. Parish, Benjamin D., B.S., M.D., Associate Professor in Otology, Graduate School of Medicine, University of Pennsylvania; Aurist and Laryngologist to St. Agnes' and Chestnut Hill Hospitals. 1927 Spruce St.

ELECTED

1899. Parke, William E., M.D., Consulting Obstetrician to the Episcopal Hospital; Associate Surgeon to the Kensington Hospital for Women; Gynecologist to the Frankford Hospital. 1534 N. Fifteenth St.

1925. Patten, Clarence A., M.D., Assistant Professor of Neurology, Graduate School of Medicine, University of Pennsylvania; Visiting Neurologist, Philadelphia General Hospital; Neurologist, Lankenau Hospital and Delaware General Hospital. 2037 DeLancey St.

1910. Patterson, Ross Vernet, M.D., Dean and Associate Professor of Medicine, Jefferson Medical College; Physician to the Philadelphia General Hospital; Physician to the Protestant Episcopal Hospital; Assistant Physician to the Jefferson Hospital. 2126 Spruce St.

*1926. Paul, John Rodman, A.B., M.D., Assistant Professor of Medicine, School of Medicine, Yale University; Associate Physician, New Haven Hospital and Dispensary. Yale University, New Haven, Conn.

1922. Pelouze, P. S., M.D., Associate in Urology, School of Medicine, University of Pennsylvania; Surgeon-in-Charge, Urological Dispensary, University Hospital. 1737 Chestnut St.

1909. Pemberton, Ralph, M.S., M.D., Visiting Physician and Director of the Department of Metabolic Diseases, Presbyterian Hospital; Associate Professor of Medicine, Graduate School, University of Pennsylvania. Paoli, Pa.

1930. Pendergrass, Eugene P., M.D., Assistant Professor of Roentgenology, School of Medicine and Assistant Professor of Radiology, Graduate School of Medicine, University of Pennsylvania; Assistant Roentgenologist, University Hospital. Ardmore, Pa.

1914. Pepper, O. H. Perry, B.S., M.D., Professor of Clinical Medicine, School of Medicine, University of Pennsylvania. University Hospital, Thirty-sixth and Spruce Sts.

†1902. Pepper, William, A.B., M.D., Dean of the School of Medicine, University of Pennsylvania; Member of the Council on Medical Education and Hospitals of the American Medical Association. University of Pennsylvania.

1916. Percival, Milton Fraser, M.D. 2332 S. Broad St.

1917. Peter, Luther C., A.M., M.D., Professor of Diseases of the Eye, Temple Medical School; Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania. 1930 Chestnut St.

1912. Petty, Orlando H., B.S., A.M., M.D., Professor of Diseases of Metabolism, Graduate School of Medicine, University of

ELECTED

Pennsylvania; Chief of the Department of Diseases of Metabolism, Hospitals of Graduate School of Medicine, University of Pennsylvania; Chief of Department of Metabolism, Philadelphia General Hospital. 1803 Pine St.

1905. Pfahler, George E., M.D., Sc.D., D.M.R.E. (Camb.), Professor of Radiology and Vice-Dean, Department of Radiology, Graduate School of Medicine, University of Pennsylvania; Director of Radiological Department, Misericordia Hospital. 1321 Spruce St.

1915. Pfeiffer, Damon B., A.B., M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon, Methodist and Abington Memorial Hospitals; Assistant Surgeon, Presbyterian and Lankenau Hospitals. 1822 Pine St.

1907. Pfromm, George W., Ph.G., M.D., Assistant Physician to the American Stomach Hospital; Consulting Physician to the German Protestant Home for the Aged. 269 S. Nineteenth St.

1907. Phillips, Horace, M.D., Assistant Surgeon, U. S. Public Health Service; Examining Physician, Office of the District Attorney. 4505 Chester Ave.

1911. Piersol, George Morris, B.S., M.D., Professor of Medicine and Vice-Dean for Medicine, Graduate School of Medicine, University of Pennsylvania; Physician to the Philadelphia General, Presbyterian and Methodist Episcopal Hospitals. 1913 Spruce St.

1917. Piper, Edmund Brown, B.S., M.D., Professor of Obstetrics, School of Medicine, University of Pennsylvania; Obstetrician to the University Hospital; Assistant Obstetrician and Gynecologist to the Philadelphia General Hospital. 2031 Locust St.

1905. Pitfield, Robert L., M.D., Pathologist to the Germantown Hospital; Bacteriologist to the Chestnut Hill Hospital for Lung Diseases. 5211 Wayne Ave.

1922. Pleasants, Henry, Jr., A.B., M.D., Assistant Visiting Physician, Chester County Hospital, West Chester, Pa.; Assistant in Neurological Clinic, Orthopedic Hospital. West Chester, Pa.

1896. Posey, William Campbell, M.D., Consulting Ophthalmic Surgeon to Wills, Howard, Chestnut Hill and Abington Hospitals. Radnor, Pa.

‡1899. Potts, Charles S., M.D., Professor of Neurology, Graduate School of Medicine, University of Pennsylvania; Neurologist to the Philadelphia General Hospital; Consulting Neurologist,

ELECTED

Lankenau Hospital; Consulting Neurologist, Hospital for the Insane, Atlantic County, New Jersey. 2018 Chestnut St.

*1907. Price, George E., M.D. Stimson Building, Seattle, Wash.

*1920. Quicksall, William E., M.D. E. 216 Fifteenth Ave., North, St. Petersburg, Fla.

1908. Radcliffe, McCluney, A.M. (Lafayette), M.D., LL.D., Consulting Ophthalmologist, Presbyterian Hospital; Consulting Surgeon to the Wills Hospital. 1930 Chestnut St.

1913. Randall, Alexander, A.M., M.D., Associate Professor of Surgery in charge of the Department of Urology, School of Medicine, University of Pennsylvania. 1323 Medical Arts Building.

1887. Randall, B. Alex., M.A., M.D., Ph.D., Emeritus Professor of Otology in the School of Medicine, University of Pennsylvania; Ear Surgeon to the Children's Hospital; Consulting Aurist to the Pennsylvania Institution for the Dear and Dumb and to Memorial Hospital, Roxborough. 2320 DeLancey St.

1926. Ravdin, I. S., B.S., M.D., J. William White Professor of Surgical Research in the School of Medicine and Assistant Professor of Research Surgery in the Graduate School of Medicine, University of Pennsylvania; Surgeon, University Hospital. 4623 Larchwood Ave.

*1904. Ravenel, Mazyck P., M.D., Professor of Preventive Medicine and Bacteriology and Director of the Public Health Laboratory at the University of Missouri. 203 Westmount Ave., Columbia, Mo.

1928. Reese, Warren S., M.D., Assistant Ophthalmologist, Pennsylvania Hospital; Assistant Surgeon, Wills Hospital; Associate Ophthalmologist, Delaware County Hospital; Associate in Ophthalmology, Graduate School of Medicine, University of Pennsylvania. 230 S. Twenty-first St.

1925. Reeves, Rufus S., B.S., M.D. 2227 Spruce St.

1920. Rehfuss, Martin E., M.D. N. E. Cor. Sixteenth and Spruce Streets.

1921. Reiff, E. Paul, A.B., M.D., Member of Medical Staff, Methodist Episcopal Hospital. 5051 Chestnut St.

1919. Reimann, Stanley P., M.D., Director of the Research Institute, Lankenau Hospital; Associate Professor of Surgical Pathology, Graduate School of Medicine, University of Pennsylvania. 516 Arbutus St., Germantown.

ELECTED

1929. de Rényi, George S., M.D., Associate Professor of Anatomy, University of Pennsylvania. 216 S. Forty-sixth St.

1923. Repplier, Sidney, J., M.D., Medical Director, Curtis Hospital, Curtis Publishing Company. Chestnut Hill, Phila.

1906. Rhein, Robert D., M.D., Chief Physician to the Clinic of the American Hospital for Diseases of the Stomach; Physician to the Philadelphia Home for Incurables; Examining Physician to the White Haven Sanatorium. S. W. Cor. Fifteenth and Pine Sts.

‡*1910. Rhoads, Samuel, M.D. Altadena, Calif.

1919. Richardson, Russell, A.M., M.D., Director of the Laboratory, Methodist Episcopal Hospital; Pathologist to Abington Memorial Hospital and the Associated Medical Clinics; Associate in Medicine, University of Pennsylvania. 320 S. Sixteenth St.

1919. Ridpath, Robert F., M.D., Professor of Rhino-laryngology, Temple University; Associate Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania; Laryngologist, Samaritan and Jewish Hospitals. 1737 Chestnut St.

1898. Riesman, David, M.D., Sc.D., Professor of Clinical Medicine, School of Medicine, University of Pennsylvania; Visiting Physician to the Philadelphia General and University Hospitals; Consulting Physician to the Jewish and Woman's Hospitals. 1520 Spruce St.

1895. Ring, G. Oram, A.M., M.D., Consulting Ophthalmologist to the Episcopal Hospital; Ophthalmologist to the Widener Memorial Home for Crippled Children; Consulting Ophthalmologist to the American Oncologic Hospital. N. E. Cor. Seventeenth and Walnut Sts.

1899. Roberts, Walter, M.D., Professor of Otology in the Graduate School of Medicine, University of Pennsylvania; Otologist to the Methodist Episcopal Hospital; Laryngologist to the Philadelphia General Hospital. 1921 Spruce St.

1903. Robertson, William Egbert, M.D., Professor of Theory and Practice of Medicine and of Clinical Medicine in Temple University; Physician to the Samaritan and Garretson Hospitals. 327 S. Seventeenth St.

‡1903. Robinson, William Duffield, Ph.G., M.D. 2012 Mount Vernon St.

1912. Rodman, John Stewart, M.D., Professor of Surgery, Woman's Medical College; Surgeon-in-Chief, Woman's College Hospital;

ELECTED

Attending Surgeon, Bryn Mawr Hospital; Assistant Surgeon to the Presbyterian Hospital. 1310 Medical Arts Building.

1928. Rose, Edward, M.D., Instructor in Medicine, University of Pennsylvania; Medical Registrar, University Hospital. 1726 Pine St.

1909. Rosenberger, Randle C., M.D., Professor of Preventive Medicine and Bacteriology in the Jefferson Medical College. 1001 Walnut St.

*1907. Royer, B. Franklin, M.D. 370 Seventh Ave., New York City.

†1905. Rugh, James Torrance, A.B., M.D., Clinical Professor of Orthopedic Surgery in the Woman's Medical College of Pennsylvania; Orthopedic Surgeon to the Jefferson Medical College; Orthopedic Surgeon to the Methodist and the Philadelphia General Hospitals. 911 Medical Arts Building.

1929. Ryan, William John, M.D., Visiting Surgeon to St. Mary's Hospital and to St. Vincent's Hospital for Women and Children. Medical Arts Building.

1905. Sartain, Paul J., A.M., M.D. 2006 Walnut St.

1908. Sautter, Albert C., M.D., Assistant in the Eye Dispensary of the Lankenau Hospital. 5909 Greene St., Germantown.

1927. Saxon, Gordon J., M.D. 1506 Medical Arts Building.

1906. Saylor, Edwin S., M.D., Consulting Ophthalmologist, American Hospital for Diseases of the Stomach. 1737 Chestnut St.

1920. Scarlett, Hunter W., M.D., Associate in Ophthalmology in the Graduate School of Medicine, and Medical Examiner in the Department of Physical Education, University of Pennsylvania. 230 S. Twenty-first St.

1917. Schaeffer, Jacob Parsons, A.M., M.D., Ph.D., Sc.D., Professor of Anatomy and Director of the Daniel Baugh Institute of Anatomy of the Jefferson Medical College. 4634 Spruce St.

1899. Schamberg, Jay F., M.D., Professor of Dermatology and Syphilology, Graduate School of Medicine, University of Pennsylvania; Director, Research Institute of Cutaneous Medicine. 1402 Spruce St.

1929. Scheffey, Lewis C., P.D., M.D., Assistant Gynecologist, Jefferson Hospital; Associate in Gynecology, Jefferson Medical College. 267 S. Nineteenth St.

1929. Schenck, Harry P., B.S., M.D., Research Fellow in Allergy and Assistant Instructor in Otolaryngology, University of Pennsylvania; Lecturer and Instructor in Pathology, Evans

ELECTED

Institute; Assistant Otolaryngologist, Children's Hospital. 1912 Spruce St.

1917. Schnabel, Truman Gross, A.B. (Lehigh), M.D., Associate in Medicine, University of Pennsylvania; Physician, Philadelphia General and Howard Hospitals, and Medical Dispensary University Hospital. 1704 Pine St.

1917. Schoff, Charles H., M.D. Media, Pa.

1928. Schofield, Frederick S., A.B., M.D., Associate in Urology, Medical School, University of Pennsylvania; Chief of Urological Dispensary, University Hospital; Urologist, Orthopedic Hospital; Assistant Urologist, Chestnut Hill Hospital. 822 Medical Arts Building.

1921. Schumann, Edward Armin, A.B., M.D., Assistant Professor of Obstetrics, School of Medicine, University of Pennsylvania; Obstetrician and Gynecologist, Philadelphia General Hospital; Gynecologist, Frankford Hospital; Lecturer on Obstetrics, Jefferson Medical College; Consulting Gynecologist, Rush Hospital. 1814 Spruce St.

1887. de Schweinitz, George Edmund, A.M., M.D., LL.D., L.H.D., Sc.D., Professor of Ophthalmology in the Graduate School of Medicine, University of Pennsylvania; Consulting Ophthalmic Surgeon to the Orthopedic, Philadelphia General and Bryn Mawr Hospitals. 1705 Walnut St.

*1913. de Schweinitz, George Lord, B.S., M.D., Assistant in the Department of Ophthalmology, St. Luke's Hospital, Bethlehem, Pa. 85 East Broad St., Bethlehem, Pa.

1910. Schwenk, Peter N. K., M.A., M.D., Attending Surgeon to Eye Department of the Pennsylvania Hospital; Attending Surgeon to the Wills Hospital. 1417 N. Broad St.

1930. Scott, John P., M.D., Associate in Pediatrics, University of Pennsylvania; Pediatrician, Presbyterian Hospital; Assistant Physician, Children's Hospital. 2040 Pine St.

1917. Service, Charles A., M.D. Bala, Pa.

1924. Shallow, Thomas A., M.D., Professor of Surgery, Jefferson Medical College. 3942 Chestnut St.

1908. Shannon, Charles E. G., A.B., M.D., Instructor in Ophthalmology in the Jefferson Medical College; Assistant in the Ophthalmological Clinic at the Jefferson Medical College Hospital; Ophthalmologist to the Seybert Institution. 1930 Chestnut St.

1928. Sharpe, John Semple, M.D. Haverford, Pa.

1928. Sharpless, Frederick Cope, A.B., M.D., Attending Physician, Bryn Mawr, Hospital. Rosemont, Pa.

ELECTED

1897. Sharpless, William T., M.D., Physician to the Chester County Hospital. West Chester, Pa.

1906. Shields, William G., M.D., Dermatologist to Germantown Hospital; Chief of Dermatological Clinic and Assistant Physician to the Jewish Hospital. 414 School Lane, Germantown.

*1908. Shoemaker, Harlan, A.B., M.D., Chief Surgeon to Los Angeles County Hospital; Chief of the Surgical Staff of the Methodist Hospital and of the Hospital of the Good Samaritan. 1930 Wilshire Building, Los Angeles, Calif.

‡†1893 Shoemaker, Harvey, M.D., Visiting Physician to the Sheltering Arms; Consulting Physician to the Southern Home for Destitute Children; Assistant Physician to the Lankenau Hospital; Physician to the Out-Patient Department of the Lankenau Hospital. 1727 Spruce St.

†1896. Shoemaker, William T., M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Clinical Professor of Ophthalmology in the Woman's Medical College of Pennsylvania; Ophthalmic Surgeon to Lankenau, Germantown and Pennsylvania Hospitals. 2017 Locust St.

1900. Shumway, Edward Adams, B.S., M.D., Ophthalmic Surgeon to the Lankenau, Children's and Oncologic Hospitals. 1737 Chestnut St.

1903. Sinclair, John Falconer, M.D., Epidemiologist, Presbyterian Hospital; Physician to the Philadelphia Orphan Asylum and to the Presbyterian Orphanage; Physician to the Home of the Merciful Saviour for Crippled Children. 4103 Walnut St.

1907. Sinkler, Francis Wharton, A.B., M.D., Physician to the Orthopedic Hospital and Infirmary for Nervous Diseases. 1226 Medical Arts Building.

1902. Siter, E. Hollingsworth, M.D., Genito-urinary Surgeon to the Philadelphia General Hospital. 2213 DeLancey St.

‡1920. Skillern, Ross Hall, M.D., Sc.D., Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania. 1811 Spruce St.

1928. Skillern, Samuel Ruff, M.D., Assistant Professor in Otolaryngology, Graduate School of Medicine, University of Pennsylvania. 1734 Pine St.

1929. Smith, Austin Thomas, M.D., Clinical Assistant, Department of Laryngology, Jefferson Hospital; Instructor in Laryngology, Jefferson Medical College. 259 S. Seventeenth St.

1927. Smyth, Calvin M., Jr., M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania;

ELECTED

Associate Attending Surgeon to the Methodist and Abington Hospitals. 2021 Spruce St.

1919. Smyth, Henry Field, M.D., Assistant Professor of Industrial Hygiene, School of Hygiene, University of Pennsylvania. Wayne, Pa.

1928. Spaeth, Edmund B., M.D., Associate in Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Instructor in Ophthalmology, School of Medicine, Temple University. 1930 Chestnut St.

1908. Speese, John, M.D., Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Children's and Presbyterian Hospitals. 133 S. Thirty-sixth St.

1897. Spiller, William G., M.D., Professor of Neurology in the University of Pennsylvania; Consulting Neurologist to the Philadelphia General Hospital. 4409 Pine St.

1929. Sponsler, Marshall B., A.B., M.D., Instructor in Otolaryngology, School of Medicine and Associate in Otolaryngology, Graduate School of Medicine, University of Pennsylvania; Assistant Otolaryngologist, Methodist Hospital; Visiting Otolaryngologist, White Haven Sanatorium. 1934 Spruce St.

1894. Stahl, B. Franklin, B.S., Ph.G., M.D. Haverford, Pa.

1909. Starbuck, J. Clinton, M.D., Physician to the Orphanage, Orphan Society of Philadelphia, at Wallingford, and to the Glen Mills School, Glen Mills, Pa. 42 E. Washington St., Media, Pa.

1927. Starr, Isaac, Jr., B.S., M.D., Assistant Professor of Clinical Pharmacology and Associate in Medicine, School of Medicine, University of Pennsylvania; Acting Physician, University Hospital. Chestnut Hill.

1912. Stauffer, Nathan Pennypacker, D.D.S., M.D., Laryngologist and Otologist to the Presbyterian Hospital; to the Philadelphia General Hospital, Tubercular Department; to the Home for Crippled Children. 1900 Rittenhouse Square.

1910. Stellwagen, Thomas C., Jr., M.D., Chief Clinical Assistant in the Out-Patient Surgical Department of the Jefferson Medical College Hospital. 220 S. Sixteenth St.

1895. Stengel, Alfred, M.D., Sc.D., Professor of Medicine in the University of Pennsylvania; Physician to the University Hospital and the Pennsylvania Hospital. University Hospital.

1901. Stevens, Arthur A., M.D., Professor of Applied Therapeutics in the University of Pennsylvania; Professor of Materia Medica, Therapeutics and Clinical Medicine in the Woman's

ELECTED

Medical College of Pennsylvania; Physician to St. Agnes' Hospital. 314 S. Sixteenth St.

1914. Stewart, Thomas S., B.S. (Univ. of Penna.), M.D. 1532 Pine St.

1925. Stokes, John H., A.B., M.D., Professor of Dermatology and Syphilology, School of Medicine, and Graduate School of Medicine, University of Pennsylvania; Attending Dermatologist, University and Philadelphia General Hospitals; Dermatologist, Hospital of the Graduate School of Medicine. 3800 Chestnut St.

1928. Stokes, Joseph, Jr., A.B., M.D., Associate in Pediatrics, and Chairman of the Pediatric Department, School of Medicine, University of Pennsylvania. 159 W. Coulter, St., Germantown.

1898. Stout, George C., M.D., Consulting Laryngologist and Aurist to the Presbyterian Hospital, the Children's Aid Society and the William Penn Charter School. 2015 Walnut St.

1923. Strecker, Edward, A., A.M., M.D., Professor of Nervous and Mental Diseases, Jefferson Medical College; Clinical Professor of Psychiatry and Mental Hygiene, Yale University Medical School; Neurologist, Pennsylvania Hospital; Medical Director of Department for Mental and Nervous Diseases, Pennsylvania Hospital. 111 N. Forty-ninth St.

1923. Stroud, William D., B.S., M.D., Physician to the Pennsylvania Hospital and Chief of Out-Patient and Heart Clinics; Associate Professor of Cardiology, Graduate School of Medicine, University of Pennsylvania; Associate in Medicine, School of Medicine, University of Pennsylvania; Physician in Charge of the Children's Heart Hospital, Wynnefield, Pa. 1011 Clinton St.

‡1923. Strouse, Frederick M., M.D., Assistant Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania. 1301 Spruce St.

1927. Sturgis, Samuel B., M.D. Ardmore, Pa.

1930. Sunderman, F. William, B.S., M.D., M.S., Ph.D., Instructor in Research Medicine, University of Pennsylvania; Chief of Metabolism Clinic and Chemist, Pennsylvania Hospital. Mayfair House, Germantown.

*1900. Swan, John M., M.D., Attending Physician, Park Avenue Hospital. 457 Park Avenue, Rochester, N. Y.

1928. Swartley, William B., M.D , Surgeon to Germantown Dispensary and Hospital and to Chestnut Hill Hospital; Demonstrator of Anatomy, Jefferson Medical College. 6002 Greene St., Germantown.

ELECTED

1900. Talley, James Ely, A.B., M.D., Professor of Cardiology, Graduate School of Medicine, University of Pennsylvania; Physician to the Presbyterian Hospital. 1811 Spruce St.

1886. Taylor, John Madison, A.B., and A.M. (Princeton), M.D., Professor of Physical Therapeutics and Dietetics, Medical Department, Temple University. 1504 Pine St.

1887. Taylor, William J., M.D., Surgeon to the Orthopedic Hospital and Infirmary for Nervous Diseases; Consulting Surgeon to St. Agnes', West Philadelphia Hospital for Women and Woman's Hospitals. 1825 Pine St.

1886. Taylor, William L., M.D. 2223 Tioga St.

‡†1910. Thomas, Benjamin A., A.B., A.M., M.D., Vice-Dean and Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Genito-urinary Surgeon, Presbyterian Hospital; Consulting Genito-urinary Surgeon, Pennsylvania Railroad and to the County Prison, Philadelphia. 1900 Spruce St.

1928. Thomas, J. Earl, M.D. Drexel Hill, Pa.

1907. Thomas, Thomas Turner, M.D., Associate Professor of Applied Anatomy and Associate in Surgery in the University of Pennsylvania; Surgeon to the Philadelphia General Hospital; Assistant Surgeon to the University Hospital. 1737 Chestnut Street.

1896. Thorington, James, A.M., M.D., Consulting Ophthalmic Surgeon to the Presbyterian Hospital; Ophthalmologist to the Ellwyn, Pa., Training School for Feeble-minded Children. 2031 Chestnut St.

1927. Thorington, J. Monroe, B.S., A.M., M.D., Instructor in Ophthalmology, University of Pennsylvania; Assistant in Ophthalmology, Presbyterian Hospital. 2031 Chestnut St.

1898. Thornton, Edward Q., M.D., Assistant Professor of Materia Medica in the Jefferson Medical College. 1331 Pine St.

1930. Thorpe, Edward S., Jr., B.S., M.D., Instructor in Pediatrics and Assistant to the Dean, School of Medicine, University of Pennsylvania; Visiting Physician to Children's and University Hospitals. 133 S. Thirty-sixth St.

1912. Torrey, Robert G., M.D., Physician to the Philadelphia General Hospital; Associate Professor of Medicine, Graduate School of Medicine, and Associate in Medicine, School of Medicine, University of Pennsylvania. 1716 Locust St.

1908. Tracy, Stephen E., M.D., Gynecologist to the Stetson Hospital; Visiting Surgeon to the Gynecean Hospital. 1930 Chestnut St.

ELECTED

1926. Tucker, Gabriel, M.D., Associate Professor of Bronchoscopy and Esophagoscopy, Graduate School of Medicine, and Associate Bronchoscopic Surgeon, University Hospital, University of Pennsylvania; Associate in Bronchoscopy and Assistant Bronchoscopist, Jefferson Medical College and Hospital; Bronchoscopist, Misericordia and Bryn Mawr Hospitals. 326 S. Nineteenth St.

*1901. Tucker, Henry, M.D. Bellevue, Md.

†1894. Tunis, Joseph Price, M.D. Chestnut Hill, Philadelphia.

1924. Turner, Charles Root, A.B., D.D.S., M.D., Dean, School of Dentistry, University of Pennsylvania. Merion, Pa.

1923. Turner, Creighton H., M.D., Associate in Medicine, Jefferson Medical College; Visiting Physician, St. Agnes' Hospital. 1731 Pine St.

1929. Tyson, Ralph M., M.D., Associate in Pediatrics, Jefferson Medical College; Pediatrist, Pennsylvania Hospital. 334 S. Twenty-first St.

1907. Ullom, Josephus Tucker, M.A., M.D., Physician to the Chestnut Hill Hospital. Carpenter and Quincy Streets, Germantown.

*1913. Vail, William Penn, B.S., M.S., M.D., Laryngologist to the Pennsylvania Institution for the Instruction of the Blind; Laryngologist to the Department for Tuberculosis of the Philadelphia General Hospital; Assistant Laryngologist and Otologist to the Out-Patient Department of the Pennsylvania Hospital; Assistant Laryngologist to the Children's Hospital. Blairstown, N. J.

1873. Van Harlingen, Arthur, Ph.B. (Yale), M.D., Emeritus Professor of Diseases of the Skin in the Philadelphia Polyclinic. Bryn Mawr, Pa.

1903. Van Pelt, William Turner, M.D. Chestnut Hill, Philadelphia.

1912. Vaux, Norris Wistar, M.D., Clinical Professor of Obstetrics, Jefferson Medical College; Chief of Maternity Service, Philadelphia Lying-in and Pennsylvania Hospitals; Surgeon to the Chestnut Hill Hospital; Surgeon to the Dispensary of the Germantown Hospital. 2031 Locust St.

*1897. Veasey, Clarence A., A.M., M.D., Ophthalmic Surgeon to the Deaconess and to St. Luke's Hospitals. Suite 404, Paulsen Building, Spokane, Wash.

†1883. Vinton, Charles Harrod, A.M., M.D. 1300 Locust St.

ELECTED

1903. Wadsworth, William Scott, M.D. 3914 Baltimore Ave.

1927. Wagers, Arthur J., M.D., Assistant Professor of Laryngology Graduate School of Medicine; University of Pennsylvania; Demonstrator of Otology and Laryngology, Jefferson Medical College; Otologist, Joseph Price Hospital. 1429 Spruce St.

1928. Wagoner, George W., M.D., Assistant Professor of Orthopedic Surgery, Graduate School of Medicine, and Associate in Pathology, School of Medicine, University of Pennsylvania; Clinical Surgeon, Orthopedic Hospital; Orthopedic Surgeon, Bryn Mawr Hospital. 2008 Walnut St.

1906. Walker, John K., M.D., Physician to the Children's Hospital of the Mary J. Drexel Home; Physician to the Out-Patient Department of the Pennsylvania Hospital. Media, Pa.

1907. Walker, Warren, M.D., Dermatologist to the Episcopal and Bryn Mawr Hospitals. 1819 Chestnut St.

1904. Walsh, Joseph, A.M., M.D., Visiting Physician to and Medical Director of the White Haven Sanatorium. Medical Arts Building.

1895. Watson, Arthur W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic and College for Graduates in Medicine; Laryngologist to the Jewish Hospital; Laryngologist and Aurist to the Mt. Sinai Hospital; Laryngologist to Home for Incurables. 2013 Locust St.

1923. Watson, William R., M.D., Chief Laryngologist, Episcopal and Diagnostic Hospitals; Assistant Chief Laryngologist, Pennsylvania Hospital. 2124 Pine St.

1903. Weber, Charles H., M.D., Assistant Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania; Consulting Physician to the Medical Dispensary, Children's Hospital; Pediatrist, Howard Hospital and to the Municipal Court. 2048 Pine St.

1930. Weeder, S. Dana, M.D., Assistant Surgeon, Germantown and Chestnut Hill Hospitals; Instructor in Anatomy, School of Medicine, University of Pennsylvania. 6110 Greene St., Germantown.

1920. Weidman, Fred. D., M.D., Professor of Dermatological Research, University of Pennsylvania; Assistant Pathologist, Philadelphia Zoölogical Garden; Attending Dermatologist, Philadelphia General Hospital. 20 Tenby Road, Llanerch, Pa.

1906. Weisenburg, Theodore H., M.D., Professor of Neuro-Psychiatry, Graduate School of Medicine, University of Pennsylvania; Neurologist, Philadelphia General Hospital,

ELECTED

and Physician to Orthopedic Hospital and Infirmary for Nervous Diseases. 1930 Chestnut St.

1923. WEISS, EDWARD, M.D., Associate in Medicine and Pathology, Jefferson Medical College; Physician to the Tuberculosis Wards, Philadelphia General Hospital. 1923 Spruce St.

1913. WELLS, P. FRAILEY, A.M., M.D., Ph.D. 754 N. Fortieth St.

1893. WESTCOTT, THOMPSON S., M.D., Consulting Pediatrist to the Methodist Episcopal Hospital; Consulting Pediatrist, Haddock Memorial Home. 1720 Pine St.

1921. WHITAKER, WILLIAM, M.D. 5448 Germantown Ave.

1901. WHITE, COURTLAND Y., M.D., Director of the Pathological Laboratories of the Episcopal Hospital; Pathologist to the Children's Hospital and to the Kensington Hospital for Women; Chief Bacteriologist to the Bureau of Health, Department of Health and Charities, Philadelphia; Associate Professor of Bacteriology in the Graduate School of Medicine, University of Pennsylvania. 6611 N. Tenth St.

*1905. WHITEWAY, HAROLD M., M.D. 2356 W. Twenty-first St., Los Angeles, Calif.

1898. WHITING, ALBERT D., M.D., Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate Surgeon, Lankenau Hospital; Surgeon, Germantown Hospital. 333 S. Eighteenth St.

1925. WIDMANN, BERNARD P., M.D., Assistant Professor of Radiology, Graduate School of Medicine, University of Pennsylvania; Chief of the Radiological Department of the Philadelphia General Hospital. 250 S. Eighteenth St.

1914. WILLARD, DE FOREST P., B.S. (Univ. of Penna.), M.D., Professor of Orthopedics, Graduate School of Medicine, University of Pennsylvania; Orthopedic Surgeon to the Graduate and Delaware Hospitals and Consultant to the Shriners' Hospital for Crippled Children. 1916 Spruce St.

1907. WILLIAMS, CARL, B.S., M.D., Ophthalmic Surgeon to the Germantown Hospital; Instructor in Ophthalmology in the University of Pennsylvania; Ophthalmic Surgeon to the Pennsylvania Institution for the Deaf and Dumb. School Lane and Greene Sts., Germantown.

1924. WILLIAMS, HORACE JAMES, M.D., Otolaryngologist to Germantown, Roxborough Memorial and Municipal Hospitals; Instructor in Otolaryngology, School of Medicine, University of Pennsylvania. 5908 Greene St., Germantown.

1920. WILLIAMS, PHILIP F., Ph.B., M.D., Assistant Professor of Obstetrics, School of Medicine and Graduate School of Medi-

ELECTED

cine, University of Pennsylvania; Gynecologist and Obstetrician to Philadelphia and Jewish Hospitals; Obstetrician, Abington Memorial Hospital. 2206 Locust St.

1927. WILLIAMSON, ERNEST G., M.D., Surgeon, Children's Hospital; Assistant House Surgeon, Pennsylvania Hospital; Assistant Surgeon, Presbyterian and Jefferson (Dispensary) Hospitals. 6353 Woodbine Ave.

1916. WILMER, HARRY B., M.D., Assistant Professor of Clinical Allergy in the Graduate School of Medicine, University of Pennsylvania; Visiting Physician to the Germantown Hospital; Associate Physician, Presbyterian Hospital; Director of the Allergic Clinic, Germantown and Presbyterian Hospitals. 138 W. Walnut Lane, Germantown.

1921. WILSON, GEORGE, M.D., Assistant Professor of Neurology, School of Medicine, University of Pennsylvania; Clinical Professor of Neurology, Women's Medical College of Pennsylvania; Neurologist, Philadelphia General, Episcopal and Abington Memorial Hospitals; Assistant Neurologist, University Hospital. 133 S. Thirty-sixth St.

1874. WILSON, JAMES CORNELIUS, A.M. (Princeton), M.D., Emeritus Professor of the Practice of Medicine and of Clinical Medicine in the Jefferson Medical College; Physician-in-Chief (Emeritus) to the Lankenau Hospital; Emeritus Physician to the Pennsylvania Hospital; Consulting Physician to the Bryn Mawr Hospital. 1509 Walnut St.

1902. WILSON, SAMUEL M., M.D. 1930 Chestnut St.

1897. WILSON, W. REYNOLDS, M.D. Villa Nova, Pa.

1904. WISTER, JAMES W., M.D., Physician to the Out-Patient Department of the Germantown Hospital. 5430 Germantown Ave.

*1901. WITMER, A. FERREE, M.D. 21 North Grand Ave., Baldwin, N. Y.

1918. WOLFERTH, CHARLES CHRISTIAN, A.B., M.D., Assistant Professor of Medicine, University of Pennsylvania; Director of the Clinic, Robinette Foundation, University Hospital; Consulting Cardiologist, Jewish Hospital. S. E. Cor. Thirty-sixth and Spruce Sts.

1893. WOOD, ALFRED C., M.D., Assistant Professor of Surgery in the University of Pennsylvania; Surgeon to the University, the Philadelphia General, St. Timothy's and the Howard Hospitals. 2035 Walnut St.

1900. WOOD, GEORGE, B., M.D., Laryngologist and Otologist, Orthopedic and Howard Hospitals; Professor of Laryn-

ELECTED

gology, Graduate School of Medicine, University of Pennsylvania. N. E. Cor. Twentieth and Chestnut Sts.

1903. Wood, Horatio C., Jr., M.D., Professor of Pharmacology and Therapeutics in the University of Pennsylvania; Professor of Materia Medica, Philadelphia College of Pharmacy and Science. 319 S. Forty-first St.

*1911. Woods, Andrew H., A.B., M.D., Professor of Psychiatry, College of Medicine, and Director of the Psychopathic Hospital, University of Iowa. 621 Summit St., Iowa City, Iowa.

†1897. Woodward, George, M.D. W. Willow Grove Ave., Chestnut Hill, Philadelphia.

1903. Worden, Charles B., A.M., M.D. Princeton, N. J.

1928. Wright, Carroll S., B.Sc., M.D., Associate Professor of Dermatology-Syphilology, Graduate School of Medicine, University of Pennsylvania; Attending Dermatologist, Philadelphia General Hospital. 1402 Spruce St.

1894. Zentmayer, William, M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Consulting Surgeon to the Wills Hospital; Consulting Ophthalmologist to the Glen Mills School and to St. Mary's Hospital. 1930 Chestnut St.

1887. Ziegler, Walter M.L., A.M., M.D. 1418 N. Seventeenth St.

1895. Zimmerman, Mason W., M.D., Consulting Ophthalmic Surgeon to the Germantown Hospital. 1518 Waverly St.

ASSOCIATE FELLOWS

(Limited to Fifty, of whom Twenty may be Foreigners)

American

ELECTED

1909. Billings, Frank, M.D., 1550 North State Parkway, Chicago, Illinois.

1893. Councilman, William T., M.D., Harvard Medical College, Boston, Massachusetts.

1909. Crile, George W., M.D., 1021 Prospect Avenue, S. E., Cleveland, Ohio.

1909. Dana, Charles Loomis, M.D., 53 West Fifty-third Street, New York City, New York.

1916. Estes, William Lawrence, M.D., South Bethlehem, Pa.

1912. McCaw, Brig.-Gen. Walter D., M.D., Assistant Surgeon-General, U. S. A., retired, Woodstock, Ulster Co., N. Y.

1906. Mayo, William J., M.D., Rochester, Minnesota.

1917. Miner, Charles H., M.D., Wilkes-Barre, Pa.

1906. Pilcher, Lewis Stephen, M.D., 489 Washington Avenue, Brooklyn, New York.

1892. Welch, William H., M.D., Johns Hopkins Hospital, Baltimore, Maryland.

Foreign

1928. Archibald, Edward, M.D., McGill University, Montreal, Canada.

1924. Hopkins, Sir F. Gowland, M.D., Biochemical Department, University of Cambridge, Cambridge, England.

1909. Macallum, Archibald B., M.A., M.B., Ph.D., Sc.D., LL.D., F.R.S., Department of Biochemistry, McGill University, Montreal, Canada.

1922. McCarrison, Colonel Robert, M.D., C.I.E., D.Sc. LL.D., F.R.C.P. (London), Honorary Physician to the King. Pasteur Institute, Coonoor, S. India.

ELECTED

1924. Macleod, John James R., M.D., M.B., D.Sc. (Hon.), LL.D., F.R.S., 43 Nanton Ave., Toronto, Canada.

1906. Myles, Sir Thomas, M.D., 33 Merion Square, W. Dublin, Ireland.

1885. Rendu, Jean, M.D., Lyons, France.

1928. Rolleston, Sir Humphry, Bart., Q.C.V.O., K.C.B., M.D., D.C.L. (Durh.), LL.D., D.G.C., "Southfield," Trumpington Road, Cambridge, England.

1908. Ross, Col. Sir Ronald, K.C.B., K.C.M.G., F.R.S., M.D., D.Sc., LL.D., Ross Institute and Hospital for Tropical Diseases, Putney Heath, London, S. W. 15, England.

NECROLOGICAL LIST

FELLOWS

TALLEYRAND D. MYERS, M.D.	December 26, 1929
CHARLES S. POTTS, M.D.	February 16, 1930
HIRAM R. LOUX, M.D.	February 27, 1930
MALCOLM S. COUNCILL, M.D.	March 23, 1930
RADCLIFFE CHESTON, M.D.	May 13, 1930
SAMUEL RHOADS, M.D.	May 24, 1930
BENJAMIN A. THOMAS, M.D.	May 29, 1930
JOSEPH M. FOX, M.D.	June 27, 1930
FREDERICK M. STROUSE, M.D.	August 4, 1930
EMORY G. ALEXANDER, M.D.	August 29, 1930
ROSS HALL SKILLERN, M.D.	September 20, 1930
JOSEPH S. NEFF, M.D.	November 4, 1930

LIST OF PRIZES AND LECTURES

THE WILLIAM F. JENKS MEMORIAL PRIZE

(Triennial)

1889	John Strahan, M.D.	Belf st (Ireland).
1895	Abram Brothers, M.D.	New York.

NOTE.—June 12, 1900, the William F. Jenks Memorial Prize Fund was transferred to the Library as the "William F. Jenks Memorial Library Fund."

ALVARENGA PRIZE OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA

(Annual)

1890	R. W. Philip, M.D.	Edinburgh (Scotland).
1891	L. Duncan Bulkley, M.D.	New York.
1892	R. H. L. Bibb, M.D.	Saltillo.
1894	G. E. de Schweinitz, M.D.	Philadelphia.
1895	Guy Hinsdale, M.D.	Philadelphia.
1897	Joseph Collins, M.D.	New York.
1898	S. A. Knopf, M.D.	New York.
1898	Robert Randolph, M.D.	Baltimore.
1900	David de Beck, M.D.	Cincinnati.
1901	George W. Crile, M.D.	Cleveland.
1903	William S. Carter, M.D.	Galveston.
1905	D. Chalmers Watson, M.D.	Edinburgh (Scotland).
1907	William Louis Chapman, M.D.	Providence.
1908	William T. Shoemaker, M.D.	Philadelphia.
1910	M. Katzenstein, M.D.	Berlin (Germany).
1911	Francis D. Patterson, M.D.	Philadelphia.
1914	H. B. Sheffield, M.D.	New York.
1915	J. E. Sweet, M.D.	Philadelphia.
1917	Wilburt C. Davison, M.D.	Baltimore.
1921	John W. Churchman, M.D.	New York.
1923	Edward P. Heller, M.D.	Kansas City, Mo.
1924	Gordon Cameron, M.B., B.S.	Victoria, Australia.
1925	Raphael Isaacs, M.D.	Boston.
1926	P. S. Pelouze, M.D. and Frederick S. Schofield, M.D.	Philadelphia.
1927	Emil Bogen, M.D.	Cincinnati, Ohio.
1928	J. Rodman Paul, M.D. and William U. McClenahan	Philadelphia.
1929	George M. Dorrance, M.D.	Philadelphia.
1930	Henry A. Harris, M.D.	London (Eng.)

NATHAN LEWIS HATFIELD PRIZE FOR ORIGINAL RESEARCH IN MEDICINE

(Triennial)

1901	Henry F. Harris, M.D.	Atlanta.
1909	Martin Henry Fischer, M.D.	Oakland.
1917	A. B. Macallum, M.D., F.R.S.	Toronto.
1919	Harvey Cushing, M.D.	Boston.
1921	William R. Nicholson, M.D.	Philadelphia.
1922	Sir Thomas Lewis, C.B.E., M.D., F.R.S.	London (Eng.).
1923	Frederick Grant Banting, M.D.,	Toronto.
1924	George J. Heuer, M.D.	Cincinnati.
1926	Knud Faber, M.D.	Copenhagen.
1928	John James R. Macleod	Toronto, Can.
1929	Simon Flexner, M.D.	New York.
1930	Franz Alexander	Berlin, Germany.

NOTE.—November 29, 1913, by Supplemental Deed of Trust, the title of this Fund was changed to "Nathan Lewis Hatfield Prize and Lectureship."

WEIR MITCHELL LECTURES

Jan. 17, 1911	Arthur R. Cushny, M.D.	London.
Mar. 30, 1911	Edmund B. Wilson, Ph.D., LL.D.	New York.
May 16, 1911	Svante Arrhenius	Stockholm.
Nov. 3, 1911	William T. Porter, M.D.	Boston.
Mar. 29, 1912	William H. Howell, M.D.	Baltimore.
Oct. 21, 1912	G. H. F. Nuttall, F.R.S., M.D.	Cambridge (Eng.).
April 4, 1913	H. P. Armsby, Ph.D., LL.D.	Pennsylvania.
Feb. 25, 1914	Harvey Cushing, M.D.	Boston.

PUBLIC LECTURES

Feb. 16, 1910	S. Weir Mitchell, M.D.	Philadelphia.
Nov. 17, 1910	Simon Flexner, M.D.	New York.
Dec. 15, 1910	William H. Welch, M.D.	Baltimore.
April 18, 1911	James G. Mumford, M.D.	Clifton Springs, N.Y.
Nov. 20, 1911	Talcott Williams, A.M., LL.D., Litt.D.	Philadelphia.
April 29, 1912	Owen Wister, A.M., LL.D.	Philadelphia.
Feb. 17, 1913	John K. Mitchell, M.D.	Philadelphia.
Feb. 15, 1916	Daniel J. McCarthy, M.D., and Walter Estell Lee, M.D.	Philadelphia.
April 15, 1916	Surgeon A. M. Fauntleroy	U. S. Navy.

MARY SCOTT NEWBOLD LECTURES

Mar. 29, 1917	A. T. McCormack, M.D.	Bowling Green, Ky.
April 4, 1919	Col. Thomas W. Salmon, M.C.,	U. S. Army.
Feb. 6, 1920	Alonzo E. Taylor, M.D.	Philadelphia.
Feb. 4, 1921	Maj.-Gen. Merritte W. Ireland	Washington.
April 20, 1921	Prof. William Romaine Newbold,	Philadelphia.
Nov. 11, 1921	Robert McCarrison, M.D., D.Sc., LL.D.	Oxford (Eng.).
Feb. 1, 1922	Hans Zinsser, M.D.	New York.
April 5, 1922	G. Canby Robinson, M.D.	Baltimore.
Dec. 6, 1922	Donald C. Balfour, M.D.	Rochester, Minn.
Mar. 7, 1923	J. J. R. Macleod, M.D.	Toronto.
April 4, 1923	Prof. Leon Asher, M.D.	Berne (Switzerl'd).
Mar. 5, 1924	Prof. A. Biedl	Prague (Czecho-Slovakia).
April 15, 1924	J. G. FitzGerald, M.D.	Toronto.
Oct. 31, 1924	John I. Hunter, M.D., and N. D. Royle, M.D.	Sydney (Australia).
Dec. 4, 1924	Willem Einthoven, M.D.	Leyden (Holland).
April 21, 1925	A. N. Richards, A.M., Ph.D.	Philadelphia.
Feb. 2, 1927	Eugene L. Opie, M.D.	Philadelphia.
Nov. 2, 1927	George R. Minot	Boston.
Mar. 22, 1928	Sir Humphry Rolleston, Bart., M.D.	Cambridge (Eng.).
Nov. 7, 1928	Ralph Pemberton, M.D.	Philadelphia.
Oct. 2, 1929	Joseph Barcroft, C.B.E., F.R.S.	Cambridge, (Eng.).
April 22, 1930	Sir Wilfred Grenfell, K.C.M.G., M.D.	Labrador.
May 7, 1930	Charles Singer, M.D., F.R.C.P.	London (Eng.).
Nov. 5. 1930	Prof. M. Weinberg	Paris (France).

MÜTTER LECTURES

1865	J. H. Packard, M.D.	Philadelphia.
1866	J. H. Packard, M.D.	Philadelphia.
1867	J. H. Packard, M.D.	Philadelphia.
1868	Harrison Allen, M.D.	Philadelphia.
1869	J. H. Brinton, M.D.	Philadelphia.
1872	J. da S. Solis Cohen, M.D.	Philadelphia.
1879	S. W. Gross, M.D.	Philadelphia.
1882	E. O. Shakespeare, M.D.	Philadelphia.
1885	H. F. Formad, M.D.	Philadelphia.
1888	O. H. Allis, M.D.	Philadelphia.
1890–1891	Roswell Park, M.D.	Buffalo.
1893–1894	De Forest Willard, M.D., and Guy Hinsdale, M.D.	Philadelphia.

1896	O. H. Allis, M.D.	Philadelphia.
1899–1900	J. B. Roberts, M.D.	Philadelphia.
1901	H. W. Cushing, M.D.	Boston.
1902	L. A. La Garde, M.D.	Washington.
1903	C. N. B. Camac, M.D.	New York.
1904	G. H. Monks, M.D.	Boston.
1905	A. O. J. Kelly, M.D.	Philadelphia.
1906	W. J. Mayo, M.D.	Rochester, Minn.
1907	J. Rogers, M.D., and S. P. Beebe, M.D.	New York.
1908	G. W. Crile, M.D.	Cleveland.
1909	H. D. Fry, M.D.	Washington.
1910	T. W. Hastings, M.D.	New York.
1911	C. F. Nassau, M.D.	Philadelphia.
1912	J. C. Bloodgood, M.D.	Baltimore.
1913	R. C. Coffey, M.D.	Portland, Ore.
1914	F. H. Albee, M.D.	New York.
1915–1916	Rudolph Matas, M.D.	New Orleans, La.
1916	Nelson M. Percy, M.D.	Chicago, Ill.
1917	Chevalier Jackson, M.D.	Philadelphia.
1919	Alexis V. Moschcowitz, M.D.	New York.
1920	J. Chalmers Da Costa, M.D.	Philadelphia.
1921	Prof. H. C. Jacobaeus	Stockholm (Swed.).
1922	James Ewing, M.D.	New York.
1923	J. Edwin Sweet, M.D.	Philadelphia.
1924	Evarts Graham, M.D.	St. Louis.
1925	Edward Archibald, M.D.	Montreal, Can.
1926	Walton Martin, M.D.	Ithaca, N. Y.
1927	E. Starr Judd, M.D.	Rochester, Minn.
1928	Herbert W. Carson, M.D.	London (Eng.).
1929	Robert B. Greenough, M.D.	Boston.
1930	Frank H. Lahey, M.D.	Boston.

WEIR MITCHELL ORATION

1919	Charles W. Burr, M.D.	Philadelphia.
1922	Charles Loomis Dana, M.D.	New York.
1925	Simon Flexner, M.D.	New York.
1928	Frederick Tilney, M.D.	New York.

MEMOIR OF CHARLES EUCHARIST DE MEDICIS SAJOUS, M.D., Sc.D., LL.D.*

By JAMES M. ANDERS, M.D.

Other people's idea of a man, said the philosopher Spinoza, is apt to be a better expression of their nature than of his. In discharging this duty tonight, this pleasant duty of commemorating a distinguished friend, the writer hopes that he may not lay himself open to the charge implied in the philosopher's words. Rather would he have his hearers say of him, in the language of Shakespeare:

> After my death I wish no other herald,
> No other speaker of my living actions
> But such an honest chronicler . . . .

Charles Eucharist de Medicis Sajous, a leading pioneer in the field of endocrinology, died April 27, 1929, at his home in Philadelphia, aged seventy-six years. He continued his literary and other activities, as well as systematic teaching of his favorite branch, endocrinology, at the University of Pennsylvania until his death. About one month before he died he informed the writer that he had quite recently begun a revision of his *Cyclopedia of Practical Medicine* a monumental work. Few physicians have ever shown the indefatigable industry and enthusiasm that he displayed throughout his long career.

Dr. Sajous was born at sea, in 1852, under the American flag near France, and he received his preliminary education in that country. He was the son of Count Charles Roustan de Medicis-Jodoigne, head of the French-Flemish family of that name in Florence, Italy, and the former Marie Pierette Cort. His father, to whose rank he succeeded by decree in 1893, died when he was only two years of age. Eight years later his mother married Mr. Charles Sajous, who was not titled.

The surname by which Dr. Sajous was known, therefore, is that of his stepfather, and was assumed in order to comply with the

* Read January 6, 1930.

inheritance laws of California. It is of interest to note that the French de Medicis-Jodoigne branch of the Medici family was created in 1606, as Princess of Royal Blood, by Henry IV of France, after his marriage to Maria de Medici (born in 1573), daughter of Prince Giovanni de Medici, who thus became Queen of France. At the time of this marriage the Franco-Flemish branch of the de Medici family, from which the subject of this memoir descended, was established. Although a descendant of royal ancestors, Dr. Sajous rarely referred to this fact.

Dr. Sajous spent several years of his boyhood in Mexico, where he became an expert horseman, and his first visit from California to the Atlantic Coast was made on horseback. He began his medical studies at the University of California, and in 1878 was graduated from Jefferson Medical College. Soon after his graduation, in 1881, he was appointed professor of anatomy and physiology at the Wagner Free Institute of Science, and in 1883 he became clinical lecturer on laryngology at Jefferson Medical College, a position he held until 1891. Dr. Sajous displayed a marked inventive faculty during these years as a laryngologist, having invented a number of instruments for use in nose and throat operations.

In 1897 Dr. Sajous was made professor of laryngology at the Medico-Chirurgical College, and Dean of the Faculty. Later, from 1910 to 1922, he was professor of applied therapeutics at Temple University, and from 1921 until his passing away held the professorship of applied endocrinology in the Medico-Chirurgical College and Hospital, Graduate School of Medicine of the University of Pennsylvania.

Dr. Sajous held office and membership in many medical and other societies, among which were the following: the American Medical Editors' Association, of which he served as President in 1903; the American Association for the Study of Internal Secretions, of which he was President in 1917, and the American Therapeutic Society, of which he was President in 1919. He was also a member of the American Philosophical Society, a Fellow of the American College of Physicians and of this College. Among other societies in which he held membership were the following: American Medical Association, the Philadelphia County Medical Society, the Pennsylvania State Medical Society and the Academy of Natural Sciences of Philadelphia.

It was eminently fitting that Dr. Sajous should be unanimously

elected first President of the American Association for the Study of the Internal Secretions, and that he should open the first scientific section of the Endocrinological Society of New York, and finally, that he should write the first book on endocrinology. To him has been appropriately given the sobriquet, the father of endocrinology in America. Unfortunately, however, endocrinology is as yet a less precise subdivision of medical science than bacteriology, chemistry or physiology.

In 1888 Dr. Sajous became the editor of the *Annual of the Universal Medical Sciences,* bringing out five volumes each year until 1896, when the work became known as Sajous' *Annual and Analytical Cyclopedia of Practical Medicine,* which, it has been said by high authority, gained world-wide reputation. In December, 1911, Dr. Sajous became the supervising editor of the *Medical Journal and Record,* holding that position until the year 1919.

Dr. Sajous was the author of several books on general medicine and on his special subject, endocrinology, which he was particularly interested in promoting. His two-volume work, *The Internal Secretions and the Principles of Medicine,* passed through ten editions. Among other works from his busy pen were *Curative Treatment of Hay Fever,* published in 1884, and *Diseases of the Nose and Throat,* in 1885.

In 1926 Dr. Sajous published a volume entitled *The Strength of Religion as Shown by Science.* This book was called forth by the moral and religious unrest of our times. In reviewing it, Dr. Charles H. Parkhurst said, "This work must appeal to the intelligence of all who concern themselves with the well-being of the individual, the family and the state."

In addition to the labors which he carried on as compiler, editor and author, Dr. Sajous contributed to medical and other leading journals frequent articles on a variety of subjects, the majority of which dealt with different aspects of endocrinology and organotherapy, many having been read before national and local medical and other organizations. The total number of such articles published was not less than eighty.

He was rich in foreign orders and decorations, among which may be mentioned American Medicine Gold Medallist in 1922; a commander of the Order of the Liberator of Venezuela (1889); a commander of St. John of Jersualem of Spain (1889); an officer of the Academy of France; a member of the Gold Medal Humane Society

of Belgium in 1890; a Knight of the Order of Leopold, Belgium (1890); a Knight of the Legion of Honor of France in 1892, and an officer of the same institution in 1922. Among the honorary degrees bestowed upon him by American Institutions were Doctor of Laws of St. Joseph's College, Philadelphia, in 1909, and Doctor of Science by Temple University in 1915.

He bore contumely and criticism of his views on endocrinology, which were liberally showered upon him by his professional colleagues until comparatively recent years, with much fortitude and patience. Often actuated by those chivalrous sentiments which characterized him, he was at such times particularly amiable with his antagonists. Fortunately, he lived to see the day when respect for his labors and theories was more widely accorded and his pioneering services to the medical profession appreciated; he saw criticism change to enthusiasm.

All authorities admit today that endocrinology is "a most significant phase of modern biology." It should be remembered that it rests upon much rigid laboratory experimentation and well-nigh dominates modern medicine. To a majority of present-day clinicians, however, it is still involved in more or less obscurity on account of the difficulty of accurately determining the balance or imbalance of the internal secretions and the disturbance of one or more of the ductless glands in the study of individual cases.

In this connection, it is piquant to recall that Dr. Sajous himself, while recognizing that a large group of complaints owe their discovery and classification to our knowledge of the internal secretions, informed us that we are still living in the dark days of endocrinology. Members of the profession who knew him intimately will share the writer's opinion that he expended unlimited, conscientious effort to advance endocrinology, endeavoring to elucidate its principles for the guidance of practitioners of medicine.

It has been claimed that, largely owing to his tireless industry and his research method of study, both in the laboratory and in the clinic, endocrinology has been placed on a firmer scientific basis in this country. Dr. Sajous believed that in endocrinology lie greater possibilities for power over life than in any other domain of modern scientific medicine, not excepting chemistry and physiology. During the last few years of his life Dr. Sajous rigidly adhered to and repeatedly expressed the view that adrenalin was of the greatest value as a stimulant in critical cases.

He stoutly contended that the adrenal cortex coöperates with the medulla in carrying on tissue oxidation. He also urged that the adrenals afford a foundation for an interpretation of pulmonary and tissue respiration. This view was at variance with that of most physiologists, although he claimed that experimental results published by them sustained his contention.

With regard to the pituitary body and its functions, Dr. Sajous was the chief exponent of the theory that endowed the organ with important function of protecting the body against certain intoxications, by enchancing the activity of the other ductless glands, notably the adrenals and thyroid, which he contended also produce a defensive hormone.

Dr. Sajous had ideals for which he made rare sacrifices. For example, during an early stage of his career, in 1891, he gave up a lucrative practice in laryngology and went to Paris, where he remained six and one-half years, devoting his time and energies to the study of endocrinology. While in Paris he acquired the research method of collecting and coördinating facts relating to the function of the ductless glands from which to formulate theories and principles. He brought back to America a mind schooled in the elements needed to produce the constructive thinker. By going to Paris he showed his willingness to learn, not believing that he was endowed with talent beyond the need of tutoring. And while his judgment appeared at times to give way to an excess of enthusiasm, be it recollected that he felt it incumbent upon himself to become acquainted with all of the facts appertaining to the deeper questions in endocrinology, before basing thereon either an opinion or theory. To such a member, the medical profession is compelled to vow respect and admiration.

In 1915 Dr. Sajous was operated upon for diverticulitis, from which convalescence was slow, occupying at least one year. In 1919 Dr. Sajous was seized with angina pectoris, which was quite severe during the first month, recurring daily. This was followed by mild attacks, which occurred at long intervals. His final illness, which lasted only a few hours, resulted from acute uremic intoxication.

On January 30, 1884, Dr. Sajous married Emma Bergner, daughter of Theodore Bergner, a well-known civil engineer. His only child, Louis Theodore, also a physician, was associated with his father in his teaching, practice and literary work. He died on January 16,

1929, just three months and a few days before the passing of his father. Thus was terminated the "nine-hundred-year-old name," of the senior branch of the de Medici family of Florentine history.

In the death of the subject of this memoir, the medical profession has lost a most worthy representative; his friends, an agreeable, intensely loyal and considerate comrade. Courteous, cultured, invested with dignity and the simplicity of a child, Dr. Sajous was, as was recently said of the late Dr. John B. Roberts, a gentleman of the old school. "Such a man might be a copy to these younger times." It has also been truly said that he had a "singularly ingratiating personality, in which the wise physician and the sympathetic friend were merged."

If time and space permitted, it would be interesting to relate some personal reminiscences that would reveal Dr. Sajous' capacity for friendship, versatility and everyday humanity. It should, however, be added that, although his later years were somewhat darkened by physical illness, when "nature and sickness did debate it at their leisure," he may well have felt that he had won all "that should accompany old age—honor, lone obedience, troops of friends."

MEMOIR OF THOMAS H. FENTON, M.D.*

By B. ALEX. RANDALL, M.D.

In the death of Dr. Fenton, on February 23, 1929, the College of Physicians has lost a most valuable Fellow, long a member of its Hall Committee and since its chairman and, *ex officio*, a member of the Council. His quiet, musical and courteous speech has not been often heard in our meetings; but he was a diligent worker, who had the affairs of the Hall Committee always well in hand and was its efficient executive during the dozen years that he was its chairman. Meanwhile he spent most of his day caring for his large private practice, was constant and zealous in his attention to his public medical positions; and as an earnest Mason supervised perhaps a dozen lodges which were under his survey. An ex-student of the Episcopal Academy, he felt a grateful debt to it, which he fittingly repaid in interest and gifts. A notable athlete, he did much for the Athletic Club of the Schulykill Navy, of which he served as president; and for the A.M.P.O. Medical Fraternity, the James Aiken Meigs Society and the Art Club, which he served for years as secretary and later president. But a single paper of his, on "Hygiene in the Philadelphia Schools," is listed in our library, but he presented many valuable papers which he too modestly refrained from printing. Long an interested member of the Philadelphia County Medical Society, he served it as president and was active almost from his graduation in the Pathological Society. Besides his many medical activities, he was an accomplished musician, early a chorister at St. Mark's P. E. Church, later a tenor at St. Clement's and an active singing member of the Orpheus Chorus, from 1882; its president in 1909–1910.

Dr. Fenton was a founder and the first president of the American Climatological Society, Society for Tropical Diseases, a fellow of the American Ophthalmological and of the International Ophthalmological Congress.

He had a ghastly shock when shot, on the night of January 15, 1921, by a footpad to whom he refused abject submission. The

* Read January 6, 1930.

bullet struck him below the left eye and passed through his tympanum, to lodge in his mastoid, from which it was removed by Drs. J. Chalmers Da Costa and S. Macuen Smith; but he bore little scar and made slight complaint of the damage to his hearing.

Dr. Fenton was an accomplished linguist, habitually using German in his home circle and maintaining his Latin and Greek, reading and writing them in correspondence with qualified friends. His reading was wide and discerning and he was at home in the English classics.

Dr. Fenton married, in 1885, Lizzie Spear Remak, daughter of Gustavus Remak, long a member and later President of the Commissioners of Fairmount Park; but lost her more than a year ago. He left two daughters, one notable for her sculpture, and one son, Powell, now living in Paris.

Of classic features and fine figure, he bore himself with stately grace, and, if latterly he stooped a little, it only befitted his gracious courtesy, which carried no suggestion of condescension. He was a charming colleague, whose companions not only enjoyed his conversation and manner, but felt honored by his acceptance of themselves.

MEMOIR OF HENRY MORRIS, M.D.

By ADDINELL HEWSON, M.D.

Dr. Henry Morris, the son of Dr. Robert Morris and Lucy Pickett (née Marshall), was born in Philadelphia, June 6, 1855, and died February 22, 1929. In the accompanying geneological chart (on his father's side) there were five Robert Morrises, and in addition he had a half-brother and has a son and grandson of the same name, and among these was the Robert Morris, the revolutionary financier, whose wife was a sister of the first Episcopal Bishop of Pennsylvania, William White; as a coincidence, his maternal grandfather was Robert Morris Marshall.

The Morris family settled in Oxford, Maryland, and owned considerable land there. The Marshalls were Virginia people with military tendencies, as James Marshall was a lieutenant in Alexander Hamilton's regiment. And Thomas Marshall was a colonel of the Virginia infantry in the Revolution and a lieutenant of Virginia volunteers in the French and Indian Wars.

The subject of this memoir attended a private school, located at Tenth and Chestnut Streets, under the competent preceptorship of the Rev. Albert Henry Barnes. Here he was prepared for Princeton, but abandoned the plan on account of his father's opposition, at that time, to his pursuing later a medical course. He entered Bryant and Stratton's Business College, graduating from that institution in 1873. Following this instruction he was able to write distinctly with both hands, beginning on one side of the paper with his left, then transferring the pen to the right hand and finishing the line; no change whatever being perceptible in the chirography.

In 1875 he began reading medicine with his father in Philadelphia; later he matriculated in the Jefferson Medical College and graduated in 1878. He was an assistant in Dr. J. M. DaCosta's clinical service at Jefferson Hospital for two years, 1878–1880; in the Surgical Dispensary, 1881–1883. He was assistant demonstrator of anatomy in 1878–1883; demonstrator in obstetrics in 1884–1888.

* Read January 6, 1930.

He was associated with the Howard Hospital in gynecology up to 1888. Immediately following his graduation he was a very successful quizmaster in anatomy, materia medica, surgery and gynecology, as medical men in the army, navy and marine hospital services can testify. He was private assistant to Prof. J. M. DaCosta, professor of medicine in Jefferson Medical College, for many years after severing his connection with Jefferson Medical College. He edited an edition of Biddle's *Materia Medica*, by the late John B. Biddle, professor of materia medica and therapeutics in Jefferson Medical College. He was the author of *Essentials of Practice of Medicine*, *Essentials of Materia Medica and Therapeutics*, and *Compendium of Gynecology*. He was elected to the medical staff of St. Joseph's Hospital in 1888 and served for several years, when he was made emeritus. In 1904 he succeeded the late Dr. W. H. Parish in the chair of anatomy in the Woman's Medical College of Pennsylvania and retired as emeritus in 1920.

He was elected treasurer of the Anatomical Board of the State of Pennsylvania in 1914, and was acting in that office at the time of his death. He served on the Mütter Museum committee as a member and chairman, and later as curator. He was a member of Philadelphia County Medical Society, American Medical Association, Pennsylvania State Medical Society, Fellow of the College of Physicians of Philadelphia; associate fellow and honorary member of the Altoona Academy of Medicine, associate fellow of the Association of Military Surgeons of the United States, by reason of his connection with the military corps of the State militia. He performed very effective service during the influenza epidemic in Chester, Pa. He was a member of the Sons of the Revolution, the Society of the Colonial Wars and the Descendants of the Colonial Governors.

The relations he maintained with all his associates were gentle, courteous and constructive.

He married Bessie Thomas (née Elliott), October 12, 1880, and leaves a son, Lieutenant-Colonel Robert Morris, U. S. A., and a daughter Grace, Mrs. P. L. Poe, of Baltimore, Maryland, and several grandchildren, and one of them Robert Morris.

He was true to his inheritance—a perfect gentleman.

HENRY MORRIS, M.D.,
b. June 6, 1855; *d.* Feb. 22, 1929.
m. Oct. 12, 1880, BESSIE T. ELLIOTT

ROBERT MORRIS, M.D. *m.* LUCY PICKETT MARSHALL
b. Dec. 12, 1802; *b.* Oct. 26, 1829;
d. June 18, 1891; *d.* May 1912;
m.———

ROBERT MORRIS *m.* ANNA SHOEMAKER (daughter of first Mayor of Philadelphia)
b. 1777;
d. 1806;
m.———

ROBERT MORRIS, Financier, *m.* MARY WHITE (sister of Bishop William White, first Episcopal Bishop of Pennsylvania)
b. 1733;
d. 1806;
m.———

ROBERT MORRIS, of Oxford, Maryland, *m.*———
b. 1711;
d. July 12, 1750;
m.———

ANDREW MORRIS *m.* MAUDLIN SIMPSON of Leicestershire, England

ROBERT MORRIS MARSHALL *m.* LUCY MARSHALL
b. Jan. 20, 1797; *b.* Aug. 15, 1796;
d. Feb. 10, 1870; *d.* Dec. 24, 1844;
m.———

JAMES MARSHALL *m.* HESTER MORRIS
b. Mar. 12, 1764; *b.* July 30, 1774;
d. Apr. 26, 1848; *d.* Apr. 18, 1816;
(Lieutenant in Alexander Hamilton's Regiment)

THOMAS MARSHALL *m.* MARY RANDOLPH KEITH
b. Apr. 2, 1730; *b.* Apr. 28, 1737;
d. June 22, 1802; *d.* Sept. 19, 1809;
(Col. in Virginia Inf. in Revolution and Lieut. in Va. Volunteers in French and Indian Wars)

JOHN MARSHALL *m.* ELIZABETH MARKAM
b. 1700; *b.* 1704;
d. Apr., 1752; *d.* 1775;

THOMAS MARSHALL *m.* MARTHA
b. 1655;
d. May, 1704;

CAPT. JOHN MARSHALL of Westmoreland Co., Virginia

ANNUAL ADDRESS OF THE PRESIDENT*

By JOHN H. GIBBON, M.D.

According to long-established custom your presiding officer will endeavor to present to you a résumé of the activities of the College during the past twelve months and offer some suggestions as to its future. It is difficult to make a simple rehearsal of facts, with which many of you are already familiar, interesting, but nevertheless there are certain details, especially those relating to our finances, to which I would ask your particular attention.

Finances. For several years this subject has been the one uppermost in the minds of those who have the interests of the College most at heart, and an endeavor has been made to put the situation clearly before the Fellowship. Our trouble began long before the present general financial depression set in and, fortunately, measures to overcome them were put in motion. A year ago, in beginning a discussion of our affairs, I said: "The College of Physicians today faces a serious financial situation." Fortunately, owing to the activities of a special Committee and the generosity of certain Fellows during the past year, much has been done to temporarily alleviate the situation, so that today it may be said our financial condition is certainly no worse than it was twelve months ago, and possibly a little better. But until we are able to sell the property at Thirteenth and Locust Streets, which is now costing us about $5000 a year, being the difference in the rent received and the taxes paid, the College will continue to face a serious financial condition. And, as explained last year, the sale of this property, at even a reasonable figure, will not increase our income sufficiently to meet our expenses. I will repeat a question and an answer made in our last address. How does the College manage to get along under the conditions described? By borrowing from a fund known as the Permanent Fund, wisely accumulated over a long period by putting aside a certain percentage of the dues. This fund, which had reached something over $80,000, had been reduced at the beginning

* Read January 5, 1931.

of last year to about $59,000 and during the year was further reduced by about $4000.

Something, however, has been done to remedy this situation. During the past two years one hundred and forty-seven Fellows have subscribed $46,910 and lay friends of the College $1250. Of this total ($48,160), $7015 was designated for the newly established Endowment Fund, the income of which alone can be used, the remaining $41,145 goes to rehabilitate the much-depleted Permanent Fund. The amount already received from these subscriptions is $27,090, of which $2665 goes to the Endowment Fund.

Through very definite and somewhat handicapping economies the withdrawals from the Permanent Fund during the past twelve months have been very materially reduced over that of recent years. These economies have seemed to the Council necessary, but they do not constitute the best way of meeting financial difficulties. Because, on the contrary, I think, most of those deeply interested in the College feel that its facilities and activities need to be improved and increased. It is only, then, by developing a substancial endowment that the future usefulness of the College can be assured. One hundred and forty-seven Fellows have given all they feel they can afford and some forty others have expressed an inability to subscribe; out of a Fellowship of five hundred and forty-six this leaves three hundred and fifty-nine who have not responded in any way to appeals. Tonight an amendment to the by-laws which would raise the dues from $30 to $50 will be presented, largely at the suggestion of a number of Fellows who felt they could not subscribe but would be willing to pay higher dues. I hope the increase will not be approved, for, I think, the increase may prove a hardship to some and because we must do something that will be of more permanent benefit.

Personally I feel very strongly that the individual Fellows of the College must solicit help from generously inclined laymen which will materially increase our endowment. I know that this is a poor time to beg money, but bequests in wills can be solicited at any time, and, oftentimes, one is willing to bequeath more than he is willing to give in his lifetime. Over twenty Fellows have not only subscribed a substantial amount, but have also made bequests to the College in their wills. This idea of getting bequests for the College is my last official suggestion.

Meetings. There were sixteen valuable papers read at the meetings of the College. There were three Mary Scott Newbold lectures: the first in April, by Sir Wilfred Grenfell; the second, in May, by Dr. Charles Singer, of London, and the third, in November, by Professor M. Weinberg, of Paris. The tenth Nathan Lewis Hatfield Lecture was given, in May, by Dr. Franz Alexander, of Berlin. The Mütter Lecture was delivered, in December, by Dr. Frank H. Lahey, of Boston. A special meeting was held on October 23, to celebrate the eighteen hundredth anniversary of the birth of Galen. The speakers on this occasion were Drs. William H. Welch, of Baltimore; Charles W. Burr, Giuseppe Franchinni, of Bolonga, and Burton Chance.

Three stated business meetings were held during the year, and at the one in January the following memoirs were read: Dr. Henry Morris, by Dr. Addinell Hewson; Dr. Thomas H. Fenton, by Dr. Alexander Randall; and Dr. Charles E deM. Sajous, by Dr. James M. Anders.

Deaths. Twelve active Fellows of the College died during the past year.

Dr. Tallyrand D. Myers, who was elected a Fellow of the College in 1896, died on December 26, 1929. Dr. Myers was for many years in the Medical Corps of the Navy, but retired about the time of his election, and, for a number of years, practised ophthalmology in Philadelphia. Through his influence was obtained $5000 for the establishment of the Wyeth Fund.

Dr. Charles S. Potts was elected a Fellow in 1899 and died on February 16, 1930. Dr. Potts was professor of neurology in the Graduate School of the University of Pennsylvania, was neurologist to the Philadelphia General Hospital and consultant neurologist to the Lankenau Hospital. Dr. Potts was a teacher of repute and the author of a very good textbook on neurology.

Dr. Hiram R. Loux was elected a Fellow of the College in 1907 and died on February 27, 1930. Dr. Loux was professor of genito-urinary surgery at the Jefferson Medical College and surgeon to the Philadelphia General Hospital. He had a very large general surgical practice, but for many years specialized in urology, in which field he had a most extensive experience.

Dr. Malcolm S. Council was elected a Fellow of the College in 1907 and died on March 23, 1930. Dr. Council was physician to the Bryn Mawr Hospital for many years. He had a very large

general practice on the Main Line, was highly respected by the profession and much beloved by his patients.

Dr. Radcliffe Cheston was elected a Fellow of the College in 1897 and died on May 13, 1930. Dr. Cheston was responsible for the development of the Chestnut Hill Hospital, to which he was the chief physician for many years and later served on its Board of Trustees. A memoir of Dr. Cheston will be prepared and read at the Business Meeting of the College in May.

Dr. Samuel Rhoads was elected a Fellow of the College in 1910 and died on May 24, 1930. At the time of his death he was non-resident Fellow and lived at Altadena, Calif. Dr. Rhoads was obliged to give up his practice and go to California because of poor health.

Dr. Benjamin A. Thomas was elected a Fellow of the College in 1910 and died on May 29, 1930. He was professor of urology at the Graduate School of the University of Pennsylvania and genito-urinary surgeon of the Presbyterian Hospital. Dr. Thomas was widely known in his specialty, contributed constantly to the literature and died at the height of his career.

Dr. Joseph M. Fox was elected a Fellow of the College in 1885 and died on June 27, 1930; a Fellow for forty-five years. He was a resident physician at the Pennsylvania Hospital, and for some time worked in the outpatient department. He gave up practicing, however, after some years.

Dr. Frederick M. Strouse was elected a Fellow of the College in 1923 and died on August 4, 1930. He was associate professor of laryngology at the Graduate School of Medicine of the University of Pennsylvania. Dr. Strouse was clerk of the Section on Otology and Laryngology of this College.

Dr. Emory G. Alexander was elected a Fellow of the College in 1913 and died on August 29, 1930. Dr. Alexander was assistant professor of surgery at the University of Pennsylvania, surgeon to the Episcopal Hospital, Saint Christopher's Hospital for Children and the Abington Hospital. His sudden death in the height of his surgical career was particularly distressing. Dr. Alexander had a pleasing personality and was a surgeon of unusual ability.

Dr. Ross Hall Skillern was elected a Fellow of the College in 1920 and died on September 30, 1930. He was professor of laryngology of the Graduate School of the University of Pennsylvania, and at one time president of the American Laryngological, Rhinological and Otological Society.

Dr. Joseph S. Neff was elected a Fellow of the College in 1886 and died November 4, 1930; forty-four years a Fellow of the College. Dr. Neff had not practised medicine for a great many years, but was always deeply interested in public health. He was the first medical director of the Jefferson Hospital and later Director of Public Health of Philadelphia.

Membership. On December 1 our Fellowship numbered 546, of which 528 were active Fellows, 10 American Associate Fellows and 8 Foreign Associate Fellows. There has been an increase of 6 in the active Fellowship in the past twelve months.

Library. The Library, the most valuable asset of the College, has, in spite of necessary curtailment of its expenditures, had an active year, 3058 volumes and 10,331 pamphlets being placed upon the shelves; 4 Incunabula have been added, raising our total to 391; there also have been valuable additions to our rare books. During the year a bequest of $5000 was received from the estate of Dr. Ziegler for the establishment of the S. Lewis Ziegler Fund, the income of which is to be used for the purchase of books. In the curtailment of our expenditures the Library has suffered more than any activity of the College, which is most unfortunate.

The Nurses' Directory has had a very hard year owing, we believe, largely to the general business depression. The amount which the Directory will be able to turn over to the Library is only about one-quarter of that of last year.

Sections. All the sections of the College have been most active during the year. Many valuable papers have been read and the attendance has been good.

Red Cross. At the request of the Southeastern Pennsylvania Chapter of the American Red Cross, and with the authority of the Council, a Committee has been appointed to formulate plans for the coöperation of the medical profession in Philadelphia and its vicinity with the Red Cross in case of any great calamity.

Today completes my three years as your presiding officer and I should deem myself ungrateful did I not take this occasion to thank the other officers of the College, the members of the Council, the members of various Committees and the individual Fellows for their support, their helpfulness and for their consideration of my shortcomings during this period. I also wish to express to the Librarian, to the Clerk and to the other employees of the College our appreciation of their conscientious and faithful service to the institution.

THE MÜTTER MUSEUM OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA*

By HARRY P. SCHENCK, M.D.
CURATOR

At the time of his retirement from the faculty of Jefferson Medical College, in 1856, Thomas Dent Mütter had completed an unique and valuable collection of anatomical and pathological specimens. Having occupied the professorship of surgery from 1841 to 1856, he had thrown his enthusiasm and energy into teaching and collecting, while others were writing and contributing to current medical literature. His popularity as a teacher made of him an impressive and inspiring figure to the young men of his generation. The success of his collecting propensities is attested by the survival of the museum which bears his name.

In December, 1858, three months before his death, Dr. Mütter signed an agreement with the College of Physicians of Philadelphia, to turn over his collection if the College would house the collection in a fireproof building. The collection sent from the Jefferson Medical College to the new building of the College of Physicians, in 1863, contained 474 bones, 215 wet preparations, 200 casts, 20 wax preparations, 8 papier-mâché models, 5 dried preparations and 4 oil and 376 water-color paintings.[1]

With this nucleus the collections of the Mütter Museum have grown through a period of sixty-five years. With the assurance of permanence the museum became the depository of exhibits of considerable historical importance. From the days of Morgan, Rush and Shippen came the quaint drug boxes, with their lacquer as fresh today as when they saw service. Here may be viewed the vesical calculi removed from the great John Marshall by the famed Dr. Physick. With the beginnings of aseptic surgery the curious carbolic atomizers of Lord Lister came into being. Finally, the

* Read January 6, 1930.

[1] John H. Gibbon: Thomas Dent Mütter, Annals of Medical History, vol. viii, No. 3, p. 237–241.

modern period is represented by the Chevalier Jackson collection of foreign bodies, some 2500 specimens removed by this master of his craft, being available for study.

For the student of instruments there is a wealth of material. He may linger over the Pompeian surgical collection or become absorbed in the disappearing breeds of devices, such as those once used for boring holes in vesical calculi and thus weakening them before crushing. Here we may study a method advocated by Gruithuisen, of Munich, and Elderton, of Scotland, about 1760.

The Kane Arctic case accompanied the first Grinnell expedition in search of Sir John Franklin in 1850–1851. It was the property of Dr. Elisha Kent Kane, Arctic explorer, who died in Havana in 1857. The case accompanied Kane on the second Grinnell expedition, which he commanded, and was as far north as latitude 80 degrees, 50 minutes.

Of a later period are the Civil War instruments, and as methods changed and technique progressed we trace the transition into the equipment of the World War.

In the field of ethnology the huge collection of skulls of all races, brought together by Hyrtl, of Vienna, holds much of interest. The museum also contains Hyrtl's famous preparations of the labyrinths of the ear of man at different periods of life, as well as those of all mammalian families. These, together with the ossicles, required fourteen years for preparation, and were first exhibited at the World's Exposition in Vienna, in 1873. Hyrtl's difficulties were somewhat minimized by the zealous coöperation of Professor Ducrotay de Blauville, who secured for him the temporal bones of the elephant, rhinoceros, hippopotamus, otaria jubata, physeter, tipuis and orca.

An amusing incident may emphasize some of the difficulties these workers faced. Since the time of the Roman emperors, no living hippopotamus had come to Europe. The anatomy of the internal organs of this animal remained unknown. Between 1830 and 1840 the French Government sent Monsieur Arnold d'Abbadic as ambassador to the King of Seboa. The Academy of Paris added to his instructions the request to transmit in a preserving fluid the body of a young hippopotamus.

This undiplomatic duty was discharged by the ambassador with great tact. The King was greatly afflicted with rheumatism of the joints and requested the ambassador to help him. In the Orient all

Franks were thought to be masters of the art of healing. Monsieur d'Abbadie enthusiastically agreed to help.

"Procure the medicine. I'll give my crown if I shall become well again."

"Not at all necessary; the medicine is found in your own land; it is the fat of a hippopotamus."

"Bring a hippopotamus," the King ordered his henchmen.

"Gently; you must not go so fast. It must be the fat of a pregnant female."

Parties of hunters were immediately sent into the savannahs and one of them actually brought in a pregnant female. d'Abbadie smeared the limbs of the King with its fat and he recovered from his pains. The fetus was pickled and sent to the Jardin des Plantes, where Blauville allowed Professor Hyrtl to take out the petrous portion of the temporal bone and replace it with a plaster cast.

The normal and abnormal relations of the vessels of the human placenta were first shown by Hyrtl by means of unexcelled corrosion preparations, which form another exhibit. The corrosion method of demonstrating the circulation in various organs was next developed by Hyrtl, and his large collection of such specimens is here displayed in as excellent a condition as they were fifty years ago, when they were brought to the Centennial Exposition.

At the most critical point of the Nation's financial situation, in 1893, it was discovered that President Cleveland would require an immediate serious surgical operation. Speed and complete secrecy were essential because of the dire political consequences which news of the operation would have brought about. The crisis of the moment was of a severity that has rarely been surpassed, and Charles Francis Adams called it "the most deep-seated financial storm in the history of the country."

Dr. R. M. O'Reilly (later surgeon-general of the United States) examined Mr. Cleveland on June 18, 1893, and found a rough spot on the roof of the mouth which was as large as a quarter dollar. It extended from the bicuspid teeth to within $\frac{1}{3}$ inch of the soft palate. There was some diseased bone. The growth was pronounced malignant by a pathologist. Dr. Joseph D. Bryant, long Cleveland's personal physician and friend, confirmed the diagnosis and advised immediate operation.

The President decided upon July 1 as the date, and on June 30 courageously set August 7 for a special session of Congress to vote

upon the Repeal Bill. Commodore Benedict's yacht was selected for the operation, and was repainted so that she would not be recognized as she steamed half-speed up Long Island Sound. A secret consultation between Bryant and Keen was held June 30 on the deserted deck of a Fall River boat tied at her dock in New York.

On July 1 at the final examination of the patient aboard the *Oneida,* Dr. Hasbrouck extracted two upper bicuspid teeth under nitrous oxide anesthesia. Bryant then made the the first incisions in the roof of the mouth. As the change was made to ether, Janeway kept a close watch upon the pulse and general condition. Practically the entire left upper jaw was rapidly removed, the hemorrhage controlled by packing. Eye injury and facial scarring were scrupulously avoided in order that the operation might not be betrayed.

On July 5 the President walked from his launch to his residence (Gray Gables) with but little apparent effort. But the doctors were not satisfied with the appearance of the wound at one point. A brief operation on July 17 consisted in removing all suspicious tissue and applying the electrocautery.

An artificial jaw of vulcanized rubber was fitted between operations by Dr. Kasson C. Gibson. This prevented visible deformity and supported the tissues perfectly. When in place the President's speech was excellent, with no alteration of quality.

Congress met in special session August 7, 1893 (the date Cleveland had boldly set before the performance of the operations), and after much debate finally repealed the Sherman Act. On October 26, 1893, the *Nation* said: "To Mr. Cleveland—and we might say, to Mr. Cleveland alone—belongs the honor of securing the passage of the Repeal Bill."

Dr. Keen concludes that, in the light of history, the decision to keep the operation a profound secret was not only wise but imperative. "What the consequences would have been had it become known at once, we can only surmise and shudder."

Mr. Cleveland died June 24, 1908, fifteen years after the operations. That he should have survived after the removal of a sarcoma of the jaw, without recurrence for so unusually long a period, was a great satisfaction to Dr. Bryant and his colleagues. This historic specimen, together with instruments used by these famous surgeons, is preserved in the Mütter Museum through the efforts and interest of Dr. W. W. Keen, one of those who carried this state secret through a period when its safety was vital to the Nation's welfare.

During the last century, the term "Siamese Twins" became a household expression, largely through the genius of Barnum. The "original" Siamese Twins, Eng and Chang, were born in Siam of Chinese parents. They were exhibited in practically every civilized country. Brought to this country, they settled in Mount Airy, North Carolina, where they died January 17, 1874, at the age of sixty-three years. They married sisters. Chang had 6 children and Eng 5—all normal. Because of frequent family quarrels, they had to agree to allow each to live with his wife, a week, alternating week after week.

A commission appointed by the College of Physicians of Philadelphia performed the autopsy. The livers, with their connecting vessels, are well preserved in the Mütter Museum, and casts of the twins, made after the autopsy, are exhibited under glass. The chair, upon which these famous twins were wont to sit when exhibited, is nearby. Many mementoes of this pair have been deposited here, including a scrapbook containing handbills and advertisements from the world over, which once was the property of Sir William Osler.

Elsewhere may be traced the development of the obstetrical forceps. The history of stethoscope manufacture is shown from the days of Laennec to the present. The stethoscope in the World War is not neglected, for this humble instrument of the clinician served its bit during the early period of the war, when it was used to detect enemy submarines by placing the bell against the hull of the vessel. After numerous seamen had lost their tympanic membranes during depth-bomb explosions the instrument was modified, and today the device is largely an outgrowth of radio construction. Nevertheless, the stethoscope was the first instrument which met the needs of the navies.

The Matthew M. Cryer Collection represents a lifetime of collecting on the part of a physician interested in dental and sinus abnormalities. Some of the rarest conditions are represented in multiple specimens. For the anatomist and clinician this collection is endless.

Probably the most interesting of the recent acquisitions is the Chevalier Jackson collection of foreign bodies removed by this pioneer. By the use of a clever filing system each specimen is accessible for study while protected by glass frames. The clinical histories and roentgenograms are available for study as long as

this branch of medical science is of importance to physician and student.

The increasing activity of the museum is due to recognition of the value of its teaching material for undergraduate and post-graduate use. Frequently writers on medical subjects find here the ideal illustrative material for which they have sought far. The key to many vexing problems of today may often be found in the exhibits of the past. The saturation point of usefulness has not been reached, but interest and utilization is steadily increasing.

Situated in a metropolis boasting of five internationally known medical schools the Museum of Mütter has a service to perform. It must bring to the student the dignity and inspiration of the past masters of the healing art. Medical history in graphic form may only be fostered by such museums. A central depository for the rarest teaching specimens may be of value to all. By progressiveness and vision the successors of Mütter, in carrying on the idea of the pathological museum, have built better than they knew. The result is a heritage from the past with unlimited possibilities for the future.

GOITER IN THE PHILADELPHIA DISTRICT*

By EDWARD ROSE, M.D.

AND

CHARLES H. FRAZIER, M.D.

The prevalence of goiter of various types in certain regions of the United States has been recognized for many years; attempts to obtain accurate information concerning its distribution are comparatively recent, however, and the acquisition of really valuable data dates only from the World War. Prior to this time a considerable number of independent local surveys had been made, chiefly among school children, but they could not be interpreted in a comprehensive way. A rich source of material was made available during the World War by examination of large numbers of drafted men and other recruits from all parts of the country. Although the incidence of goiter was less among these men than among children, in general the figures are proportionate for distribution, and up to the present time the Army figures represent the most complete information concerning its nation-wide occurrence. In this tabulation, Pennsylvania stood twentieth among the states with 829 cases, or 4.1 per 1000. More recently all available information has been correlated through the painstaking efforts of Olesen and his coworkers, of the Public Health Service.

Large areas still remain entirely unsurveyed, and our knowledge of goiter distribution is further handicapped by the fact that the incidence of thyroid enlargement varies widely inside many of the states represented in the Army examinations. This is true of Pennsylvania. It is known in a general way that goiter is more or less prevalent in certain districts, such as the mountainous eastern central area, portions of Berks County, and the northwestern part: the actual statistical information which we have been able to collect, however, is limited to the following table, taken from Olesen:

* Read February 5, 1930.

TABLE I.

Place.	No. of children examined. M.	No. of children examined. F.	Per cent with goiter. M.	Per cent with goiter. F.
Bradford (prevalent in a small way)				
Bradford	..	280	..	9.3
Bradford	..	563	..	6.0
Erie	11,401	11,702	2.4	9.8
Erie	..	..	..	25.0
Erie	11,019	11,372	1.6	10.3
New Castle (quite prevalent)				
Pittsburgh	43,555	54,218	33.16	50.92
State—Total per cent	..	..	..	20–30%

No data concerning goiter in the Philadelphia district are available. Largely because of this fact, we have made a statistical analysis of 635 cases of thyroid enlargement which have been examined in the Outpatient Department of the Thyroid Clinic of the Hospital of the University of Pennsylvania. Although some 1200 patients have been received in this department since its inception in 1926, many of them were patients referred for follow-up examination from the Surgical Division and are not included because they represent a selected group, chiefly hyperthyroid states. It should be emphasized that this communication does not represent in any sense a goiter survey of this region of Pennsylvania and contiguous states; it is intended only to show the types of thyroid disease encountered clinically in this area, with conclusions deducible from them. A complete goiter survey is an extensive and costly procedure, best conducted by municipal or state government; such an undertaking seems highly desirable in this state, or at least in this city, and might well bring to light facts completely divergent from those which we will present.

In order to facilitate our analysis, we have arbitrarily divided the cases with hyperthyroidism into hyperplastic toxic (exophthalmic) and toxic adenomatous, and mixed types representing a combination of the two; the simple or nontoxic have been divided into adolescent, colloid and adenomatous, with a group of mixed types; and a miscellaneous group includes a few cases of cretinism without goiter, thyroiditis of various types, and carcinoma.

The toxic adenoma, so-called, has been the term applied to those firm, irregular or nodular goiters associated with hyperthyroidism, and the hyperplastic toxic group includes those cases commonly referred to as Graves' disease or exophthalmic goiter. This division

has been made with full recognition of the difference of opinion regarding the separation of hyperthyroidism into these two groups. That group which we have designated simple adolescent includes patients up to the age of thirty years, with soft, uniform, nontoxic thyroid enlargements of slight to moderate degree, and with a history of onset prior to the age of twenty years. The term simple colloid has been applied to nontoxic, smooth, uniform or lobulated goiters of fairly firm consistency, usually beginning in adult life. The simple adenomatous group includes firm, nodular goiters occurring at any age without evidence of thyrotoxicosis. This classification has been made for convenience, and does not take cognizance of several other subdivisions of simple goiter which are in common use, such as the fetal adenoma, parenchymatous, vascular, etc. It should be remembered that our diagnoses are in most instances purely clinical ones, and only confirmed by histological study in those cases which came to operation, so that there is a personal equation. Sometimes differentiation between the adolescent and colloid types is very difficult, and the distinction may be purely an artificial one.

The 635 cases were divided into 54 males and 581 females.

From our analysis the following facts may be determined:

1. Females predominate in the proportion of 10.7 to 1; in the simple group the same ratio obtains; in the toxic group they were 5.8 to 1.

2. The largest single group is that of the simple adolescent with colloid, hyperplastic toxic, simple adenomatous, and toxic adenomatous, following in the order named.

3. The incidence of toxic cases reaches its peak in the third decade, with a steady decline thereafter; the peak of incidence of simple cases occurs in the second decade.

4. The incidence of toxic adenomatous cases is only beginning to rise in that decade, (the third), which sees the hyperplastic toxic group at its height: the former group reaches its maximum in an age group which shows a sharp drop in the latter.

5. All curves of age incidence are regular in rise and fall.

6. The average age of onset from the third decade onward shows a progressive drop below the minimal limit of the age group studied.

A predominance of cases residing in and immediately about the city is to be expected. It is of some interest to note that 143, or 42 per cent of the nontoxic cases, and 56 or 39 per cent of the toxic

cases, residing in Philadelphia were born outside the district, while only 14 or 11 per cent of all the cases (toxic and simple) residing in the metropolitan area, but outside of Philadelphia, were born in remote communities.

Considerable divergence of opinion still exists as to the importance of the part played by focal infection in the production of all types of goiter. The majority of opinion apparently leans toward the view that its rôle is of, at most, doubtful importance, and this view is supported by our findings. Definite evidence of focal infection, usually in the teeth or tonsils, was present in 44 of 212 toxic cases and 65 of 423 nontoxic cases. In those cases where such foci were removed, improvement in symptoms or decrease in size of the struma were scarcely striking.

Lues was found in 1.8 per cent of the toxic, and 1.9 per cent of the simple group, but no etiological relationship could be established.

Pregnancy appeared to play a more important part in the production or aggravation of all types than might have been suspected. Nine and nine-tenths per cent of the toxic and 9 per cent of the nontoxic group of women stated that pregnancy either immediately preceded the onset of symptoms or markedly increased them. Slight thyroid enlargements are quite common in pregnancy, but our figures indicate an unusually marked relationship between the latter condition and hyperthyroidism.

The results of basal metabolism tests, together with suggestive physical findings, indicate that varying degrees of thyroid hypofunction are not uncommonly associated with nontoxic goiter. In our series of nontoxic cases, constant basal metabolism readings below − 10 per cent were found in 5 per cent of the colloid group, 11 per cent of the adolescent and 1.5 per cent of the simple adenomatous type. In many instances the dry coarse skin and hair, and general sluggishness, characteristic of hypothyroidism, were present. This association between hypothyroidism and goiter is especially important from a therapeutic standpoint, as these patients often do far better on appropriate combined doses of thyroid substance (or thyroxine) and iodine, than on iodine alone.

Definite evidence of associated disorder of other endocrines was found in only a small group (1.4 per cent) of the entire series; the principal manifestations were obesity, acromegaly and marked disorders of menstrual function. The amenorrhea and scanty menses so commonly encountered in hyperthyroidism have not been considered in this classification.

A large number of miscellaneous complicating conditions, most of them entirely coincidental, was encountered in the series. The only one to which we wish to draw attention is splenomegaly. This has been reported as occasionally occurring in hyperthyroidism; it was present in 2 patients with hyperplastic toxic goiters, and in 1 was accompanied by an eosinophilia of 14 per cent, for which no cause other than the thyrotoxicosis could be discovered; following operation the spleen gradually decreased in size, and the eosinophilia disappeared in about six months.

The amount of iodine available to the organism is an important factor in determining the size and physiological state of the thyroid gland. It is by no means the only factor, however; increased demands for thyroid secretion associated with puberty, pregnancy, lactation and the menopause; certain infections, and diets containing mainly fat and protein, also may play an important part.

The relationship between the iodine content of food and drinking water in a given locality, and the incidence of goiter in that locality, has been conclusively shown; the iodine content of water, soil and vegetables has been found lower in goitrous than in nongoitrous districts. The lack of iodine in the water supply has been compensated for artificially in some goitrous regions, with brilliant prophylactic results, as in Rochester, N. Y. Another measure employed has been the use of iodized salt, a practice borrowed from the Swiss. This has unfortunately been exploited commercially through widespread advertising by salt companies, so that at the present time iodized salt is in wide use by the American public. Any criticism of the unrestricted use of iodized salt in this region which we may make does not indicate a disregard of its great value in the prevention of goiter in those parts of the country where it is endemic. Its worth has been proven by the prophylactic results obtained from its use in such areas as Michigan and other middle western states. Most of the iodized salt used in this country contains 0.02 per cent of potassium iodide, which is more than the content of that used in Switzerland. Four grams of salt (an average daily intake) would contain about 0.65 mg. of iodine. Marine estimates that 50 mg. of iodine a year, properly distributed, is enough to maintain the thyroid in a normal state, and that the iodine storage capacity of the normal gland is about 25 mg. In view of this, the amount ingested in iodized salt by the average individual would certainly be in excess of that required. The

wisdom of its uncontrolled use in nongoitrous regions is open to serious question, in view of the facts that (1) it is impossible to accurately regulate the amount of iodine ingested by the individual; (2) that hyperthyroidism occasionally results from the use of iodine by persons without goiter, and (3) that the use of iodine in the presence of nontoxic thyroid enlargements may result in activation of the goiter.

Little seems to be known concerning the iodine content of the Philadelphia water supply. The only figures available are those of F. E. Daniels, of the State Department of Health, who in 1926 found 0.1 part per 1,000,000,000 in a Schuylkill River sample, and 0.15 parts per 1,000,000,000 in a Delaware River sample taken at Chester, part of which was Schuylkill water. If we accept McClendon's figure of 0.23 parts per 1,000,000,000 as representing the point of demarcation between iodine-poor and iodine-rich water, the evidence suggests that our water supply is deficient in iodine. Certainly, it seems that further investigation is indicated.

Even though the iodine content of a community's water supply be low, it does not follow that the population does not receive sufficient iodine; animal and vegetable foods may furnish a rich supply. This is especially true of river and marine fish and crustaceans. The following tables will convey an idea of the iodine content of certain fish and vegetables, as well as of the water supply of various communities in the State.

TABLE II.

	Mg. I per kilo. (fresh).
Clams	1.37
Oysters	1.16
Lobster	1.38
Bluefish	0.26
Cod	0.24
Mackerel	14–40

TABLE III.

Supply.	Source.	Pts. per billion.
Philadelphia	Schuylkill	0.1
Chester	Delaware	0.5
Bethlehem	Well and Lehigh	2.5
Allentown	Wells	2.4
Reading	Surface	1.5
Sunbury	Surface	0.6
S. Pittsburgh	Monon.	0.5
Erie	Lake Erie	0.6
Sharon	Shenango	0.4

TABLE IV.

Mg. I per 1000 kilos.

	Nongoitrous regions.		Goitrous regions.
Wheat	4–9.3	Wheat	1–6
Oats	23–175	Apples	3.0
Salmon	45–374	Skim milk	12
Carrots	170	Carrots	2.3

In summary then, the following conclusions are justified:

1. Accurate information is lacking concerning the distribution of goiter in the state of Pennsylvania, and particularly in the Philadelphia district.

2. A comprehensive goiter survey under the auspices of the state or city government is needed.

3. Statistical analysis of 635 cases of goiter as seen in a thyroid clinic suggests that the condition is more prevalent in the Philadelphia metropolitan district than has previously been suspected, although this cannot justifiably be considered an endemic goiter area. Non-toxic adolescent and colloid types apparently predominate, and occur chiefly before the fourth decade of life.

4. Pregnancy appeared to play an important part in the inception and aggravation of both simple and toxic varieties in this series.

5. The occurrence of thyroid hypofunction in a certain percentage of nontoxic goiters is of considerable importance, particularly from a therapeutic standpoint.

6. Available data concerning the iodine content of the Philadelphia water supply are meager, but suggest that it may be low. Further investigation of this question is needed.

CONGENITAL INSUFFICIENCY OF THE PALATE*

By GEORGE M. DORRANCE, M.D.

Several years ago I was confronted with a case of congenital short palate, a condition for which there was no satisfactory method of treatment. Congenital insufficiency and its counterpart, the rigid short palate with deficient speech, which occurs at times after cleft-palate operations, are from the standpoint of treatment exactly similar.

Fig. 1.—Artist's presentation of case with congenital shortening of the palate.

This stimulated me to make a complete survey of all the procedures employed for correcting this defect. I was dissatisfied with

* Read February 5, 1930, as a summary of an essay which won the Alvarenga Prize for 1929. The essay was published in the *Archives of Surgery*, August, 1930, vol. xxi, pp. 185–248.

the methods employed, because no operation appeared applicable in every case and many of these operations were not only useless but frequently did harm.

Anatomical studies were undertaken and various operative procedures were tried out for lengthening the velum. This resulted finally in a procedure which we believe solves the problem.

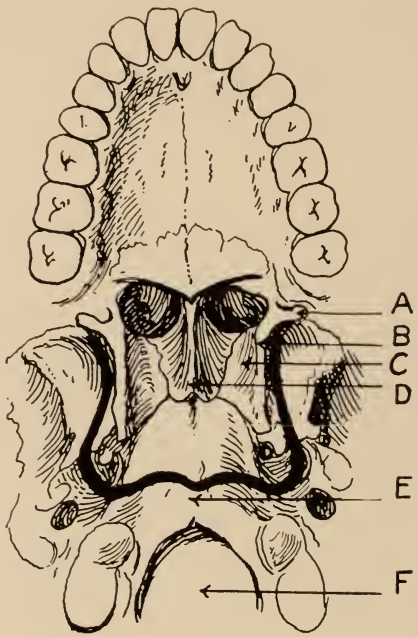


Fig. 2.—The heavy line shows the attachment of the fascia pharyngobasilaris to the base of the skull. *A*, indicates the hamular process; *B*, pterygoid plate; *C* sphenoid bone; *D*, vomer; *E*, occipital bone; *F*, foramen magnum.

Congenital insufficiency of the palate is a condition in which the velum assisted by the superior constrictor muscle of the pharynx fails to produce sphincterlike closure between the oropharynx and the nasopharynx, a function essential for the production of normal speech.

While congenital insufficiency of the palate has probably existed from the beginning of the human race, no reference in the literature occurs until 1825, when Joseph Philibert Roux called attention to the subject.

The first reference in American literature to this condition was made by Dr. J. Ewing Mears, of Philadelphia, in 1893, when he reported a case of a young woman who suffered from a marked shortening of the velum.

The term "submucous cleft palate" was introduced into the literature, in 1910, by A. Brown Kelly, of London, England. Kelly made a thorough investigation of this congenital malformation and published the first complete and systematic English treatise dealing with this subject.

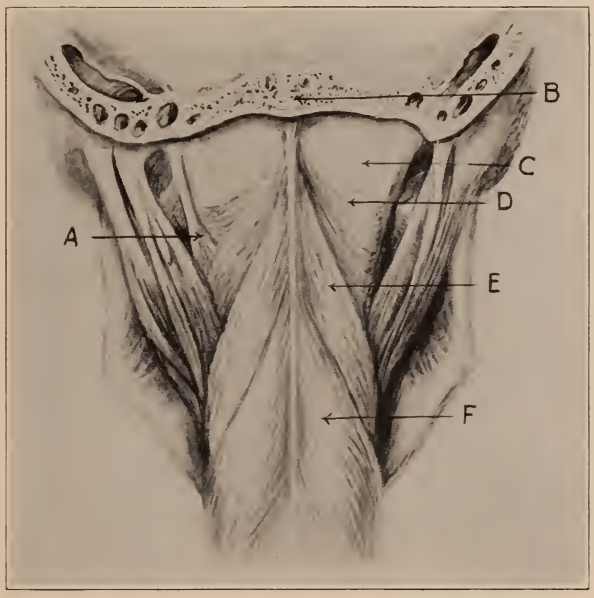


FIG. 3.—View of the pharynx from behind. *A*, indicates the pterygopharyngeus muscle; *B*, occipital bone; *C*, fascia pharyngobasilaris; *D*, superior constrictor muscle; *E*, middle constrictor muscle; *F*, inferior constrictor muscle.

That the subject of insufficiency of the palate has attracted the attention of numerous observers is evidenced by the fact that mention of this condition has been made by every important worker in the field of cleft palate.

The same conditions which cause cleft palate, namely, arrest of development due to some abnormal disturbance, may result in congenital insufficiency of the palate. Just what these factors are

which produce these conditions, we are unable to say. We know that heredity plays an important rôle, for defects may be handed down from generation to generation.

Reviewing briefly some anatomical and physiological facts, we recognize that the pharynx is a muscular tube formed essentially by the constrictor muscles and suspended from the base of the skull by the fascia pharyngobasilaris, the attachment of which is shown in Figs. 2 and 3.

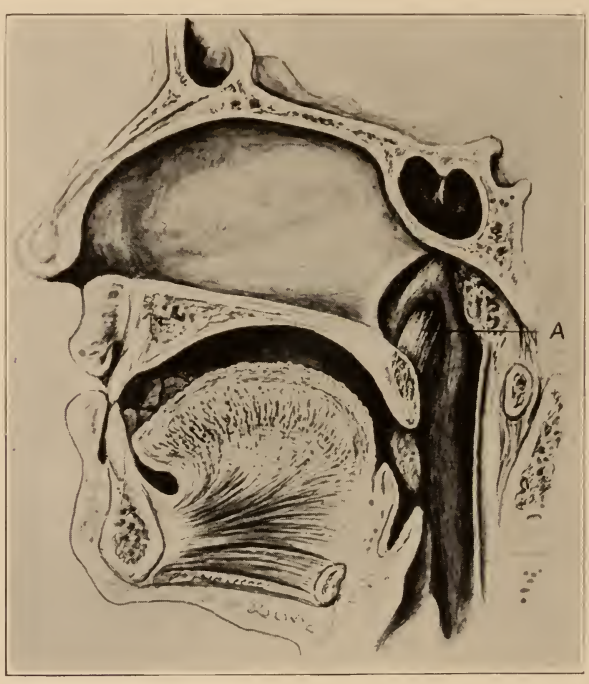


FIG. 4.—Frozen sagittal section with the levator palati muscle dissected. *A*, indicates the levator palati muscle.

It is also held dorsally to the tissue lying upon the vertebral column by fascial strands extending from the prevertebral fascia. The pharyngeal canal moves upward and downward in a wavelike manner while performing its function.

Velopharyngeal closure is accomplished by a complicated sphincter-like mechanism located at the lowermost part of the nasopharynx, (Fig. 4).

The structures concerned in causing the closure are the lowermost portion of the nasopharyngeal wall on one side and the velum on the other.

The posterior wall of the nasopharynx where velopharyngeal closure occurs, is formed by the pterygopharyngeus portion of the superior constrictor muscle, which on contraction constitutes what is usually referred to as Passavant's cushion, (Figs. 5 and 6).

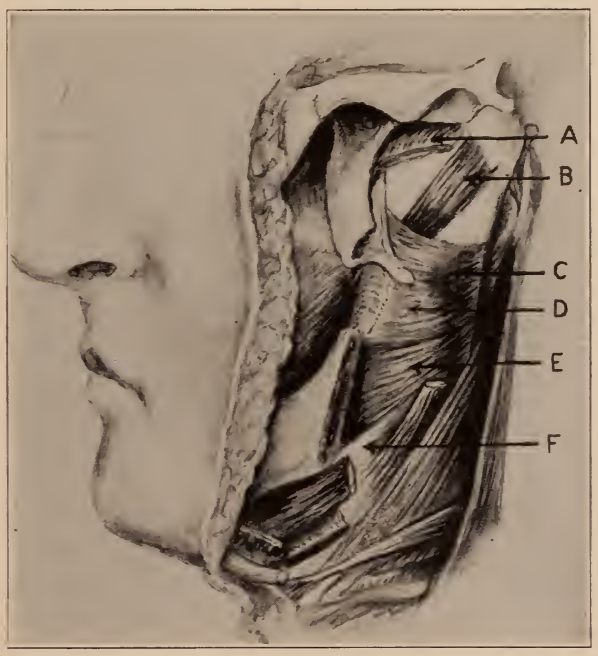


FIG. 5.—Lateral view of the pharynx, showing the superior constrictor muscle. *A*, indicates the tensor palati muscle "cut;" *B*, levator palati muscle; *C*, pterygopharyngeus; *D*, buccopharyngeus; *E*, mylopharyngeus; *F*, glossopharyngeus.

The velum from the standpoint of function is divided into two parts: an anterior horizontal portion, made up essentially of muscular tissue, and a posterior vertical portion, (Fig. 7).

At the junction of these two portions there is an interlacing of the palatal muscles where the levator palati muscles are attached to the palatine aponeurosis. The levators must be considered as acting as one muscle. The extent of backward and upward elevation

of the velum depends exclusively upon the position of insertion of the levator palati muscles.

The nasal surface of the velum, corresponding with the attachment of the levator palati muscles, approximates Passavant's cushion during velopharyngeal closure and forms the anterior segment of this mechanism. During velopharyngeal closure the uvula contracts and shortens, to make the point of insertion of the levators thicker and broader, as in Fig. 8.

FIG. 6.—View of a case of cleft velum while the patient attempts to contract the muscles slightly. Note the well-marked Passavant's cushion.

A portion of the tensor palati muscle arises from the scaphoid fossa at the base of the mesial pterygoid plate, and the spina angularis of the sphenoid bone passes around the hamular process, radiating mesialward into a fanshaped expansion, to be inserted into the palatine aponeurosis and to interlace with its fellow of the opposite side, (Figs. 9 and 10).

In the normal palate this portion of the muscle makes the palatine aponeurosis tense. In patients with congenital short palate the

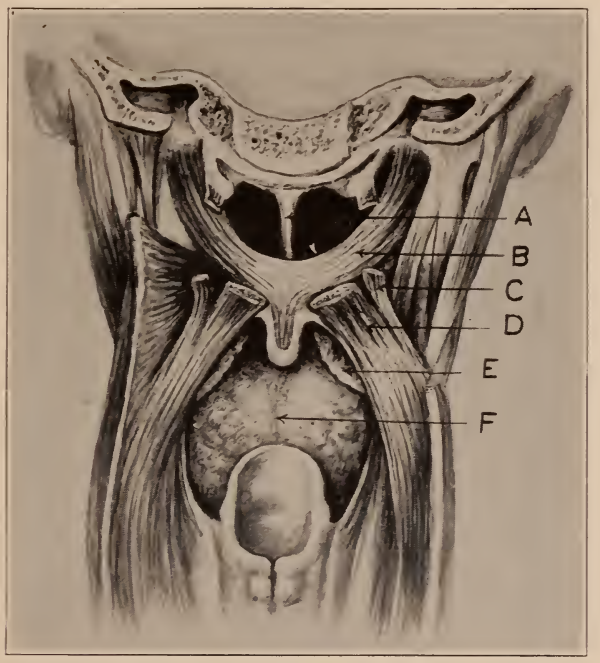


FIG. 7.—View of the palate from behind to show the levator palati muscles. *A*, indicates the septum naris; *B*, levator palati; *C*, salpingopharyngeus "cut;" *D*, palatopharyngeus "cut;" *E*, faucial tonsils; *F*, dorsum linguæ.

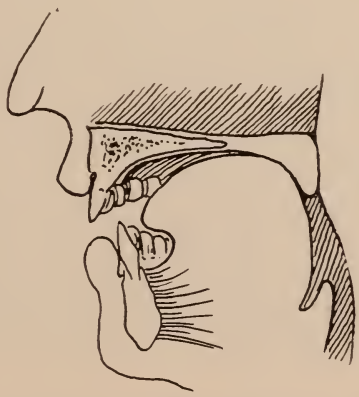


FIG. 8.—To show how normal velopharyngeal closure occurs. Note the bulging of Passavant's cushion and the contraction of the vertical portion of the velum which aids in the sphincteric action.

tensor palati muscles act separately and pull the velum too far forward and outward. The point of insertion of the levator palati muscles in these cases is displaced forward by the action of the

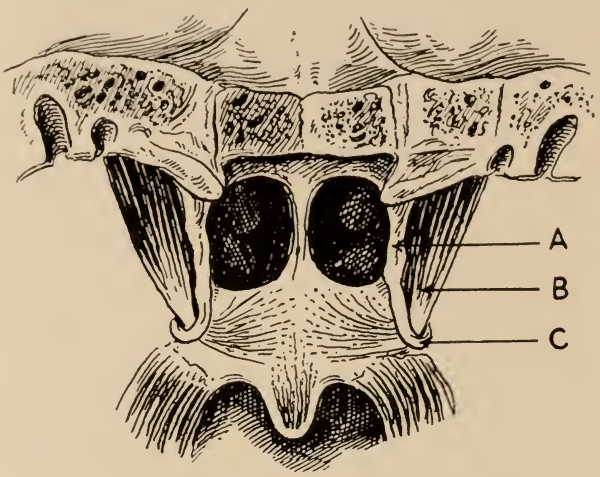


Fig. 9.—Sketch showing the tensor palati muscles. *A*, indicates the mesial pterygoid plate; *B*, tensor palati muscle; *C*, hamular process.

Fig. 10.—Photograph of specimen from the Mütter Museum of the College of Physicians of Philadelphia, showing dissection of the velum in man by Dr. Adam Politzer. *A*, indicates levator palati; *B*, palatine aponeurosis; *C*, tensor palati; *D*, hamular process.

tensor palati muscles, hence the levators are unable to raise the velum upward and backward to the desired point against Passavant's cushion.

To counteract the forward and outward pull of the tensor palati muscles in cases of congenital short palate, it is necessary to divide the posterior portion of the tendon of the tensor palati muscles.

When one observes velopharyngeal closure through the pharyngoscope or in individuals who have suffered the loss of the nose and the septum naris, the nasopharynx resembles a funnel, the apex of which is directed downward. As closure occurs, the walls of the pharynx are pulled forward and inward by the superior constrictor muscles. The longitudinal bands formed by the salpingopharyngeus muscles contract, thus increasing the bulk and thickness of the walls and narrowing the lumen. The anterior segment is drawn upward and backward by the levator palati muscles.

Velopharyngeal closure is complete in normal individuals during the acts of swallowing, gargling, whistling or in the pronunciation of certain letter sounds which are free from nasal intonation.

The diagnosis of congenital shortening of the palate can only be definitely determined after the child makes efforts to speak. Children with congenital shortening of the palate usually learn to speak at a later period than normal children. These children speak very indistinctly, due to faulty velopharyngeal closure. It is true that as these children grow older and become more persistent in their efforts to speak, their speech improves, and this improvement depends on their ability to develop closure of the anterior naris to compensate for this velopharyngeal insufficiency. They develop movements of the facial muscles, especially the compressor naris and corrugator superciliary. These patients are referred to as "face talkers."

The final diagnosis is made from the following symptoms:

1. Nasalizing (a condition which begins at the period when the patient began to talk). The vowels take a nasal tone, *i. e.*, the letter sound "Ah" becomes "An," etc. All the consonants become altered with the exception of the letters "M" and "N," which retain their characteristic sounds. Sigmatismus, or inability to produce the letter sound "S," becomes very pronounced in these cases.

2. These patients suffer from shortness of breath in speaking, due to the loss of air through the nose.

3. They are unable to whistle, hiss or to blow out a candle flame from a distance.

4. Intraoral examination reveals a distinct velopharyngeal insufficiency, and palpation may reveal a submucous cleft of the hard palate. This submucous cleft may vary from a mere notch to a considerable V-shaped deficiency in the median portion of the bony palate, (Fig. 11).

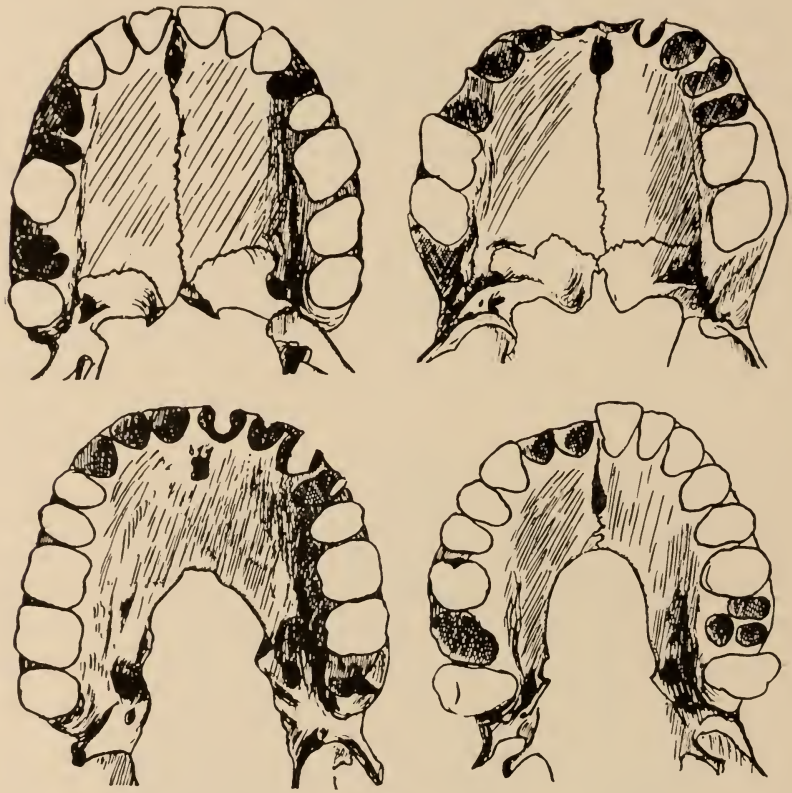


FIG. 11.—To illustrate extent of deficiency in the hard palate. (From A. Brown Kelly.)

The palatine mucosa is found intact. The submucous cleft may involve the velum alone.

If the palate, assisted by the superior constrictor muscle of the pharynx, is unable to shut off the nasopharynx, it must be considered a short palate. While complete velopharyngeal closure is essential

for normal function, this opening is not always hermetically closed during normal speech, since a certain amount of air escapes through the nostrils as certain letter sounds are pronounced. The shortness of the palate determines the degree of insufficiency. A reliable test is to hold a piece of cotton in front of the anterior nares while the patient makes an effort to produce a nonnasal letter sound, such as "Ah." Any air escaping causes the cotton to move and shows that the closure is incomplete.

In reviewing the literature, one is constantly impressed by the fact that the early pioneers of palatal surgery recognized that the defect in speech in cases of congenital insufficiency of the palate, or cleft palate, is due to an insufficient velopharyngeal closure, the result of a shortened velum. Many ingenious methods were adopted to remedy this insufficiency. These procedures may be grouped under three general headings.

Fig. 12.—Passavant's studshaped obturator. This is placed into a button-shaped incision made transversely into the velum. The object is to displace the velum backward to meet the posterior wall of the pharynx. (After Küster.)

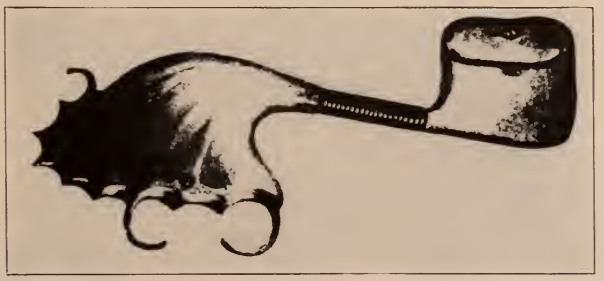


Fig. 13.—The Schlitsky-Wolff obturator, to help in producing velopharyngeal closure. (After Partsch.)

1. *The use of prosthetic appliances in the form of obturator-vela.*

Many surgeons, after failing to obtain good functional results from operations, advise the use of an artificial velum. Most patients with congenital shortening of the palate cannot tolerate contrivances in the mouth. Irritation is produced, spasms occur and frequent alterations of the apparatus must be made to obtain any sort of a result.

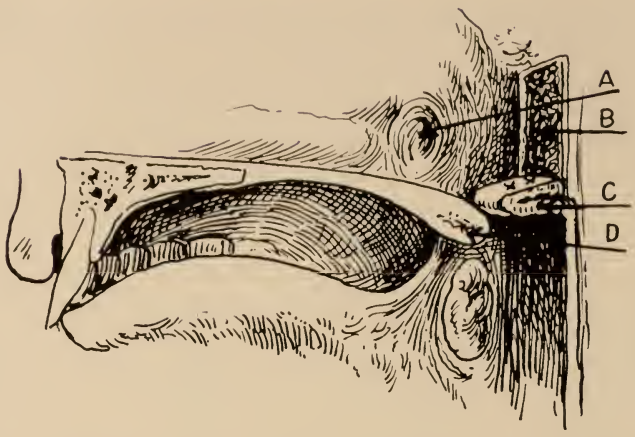


FIG. 14.—Passavant's method of making a shelflike projection upon the posterior pharyngeal wall to produce velopharyngeal closure. *A*, indicates the Eustachian tube; *B*, denuded area; *C*, projection; *D*, velum.

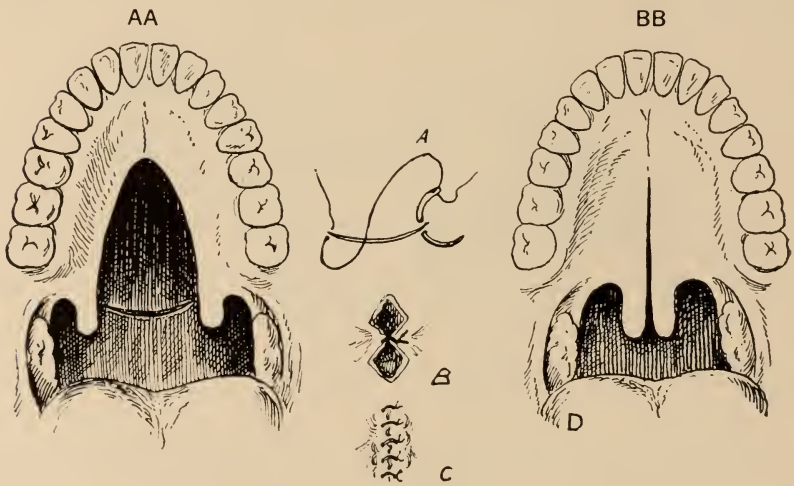
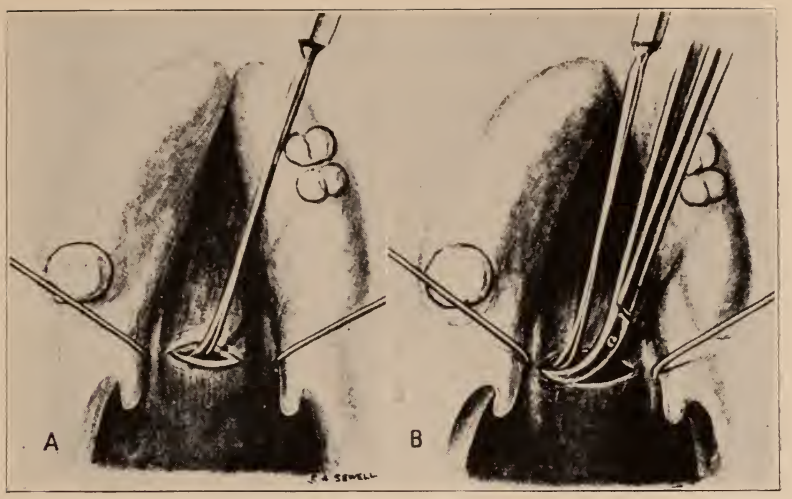
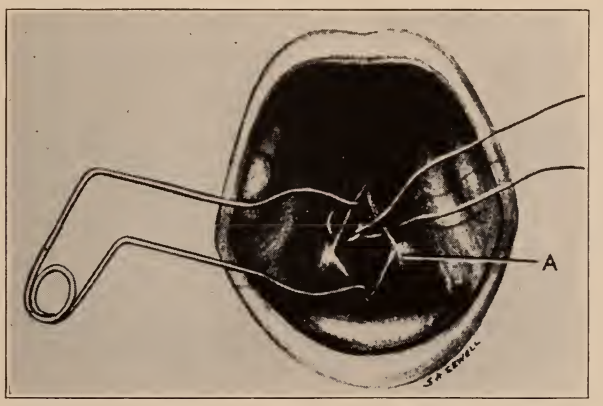


FIG. 15.—Rutenburg's method of narrowing the lumen of the pharyngeal canal. *AA*, shows horizontal incision made in the pharyngeal wall; *A*, shows application of suture at the extremities of the incision to produce vertical suture; *B*, shows the wound after knotting the suture; *C*, shows the incision completely sutured in the vertical direction; *BB*, shows how the pharyngeal canal has been narrowed. Note how the sides of the cleft in the velum have approximated without any lateral incisions.





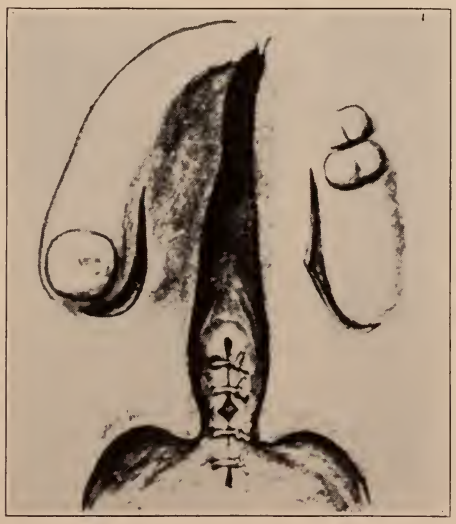


Fig. 16.—To illustrate Wardill's operation for narrowing the lumen of the pharyngeal canal. The incision on the posterior pharyngeal wall extends transversely between the salpingopharyngeal bands of muscle tissue. (From Wardill.)

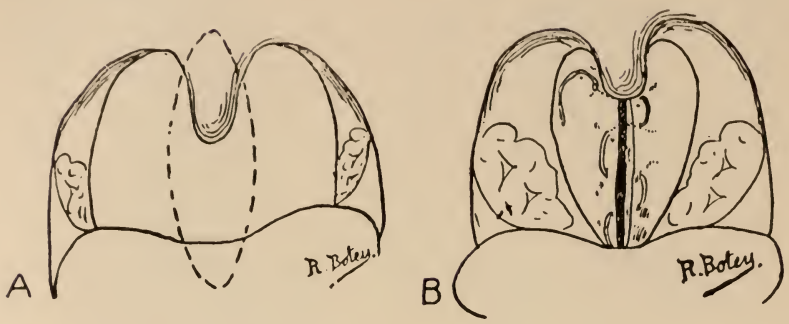
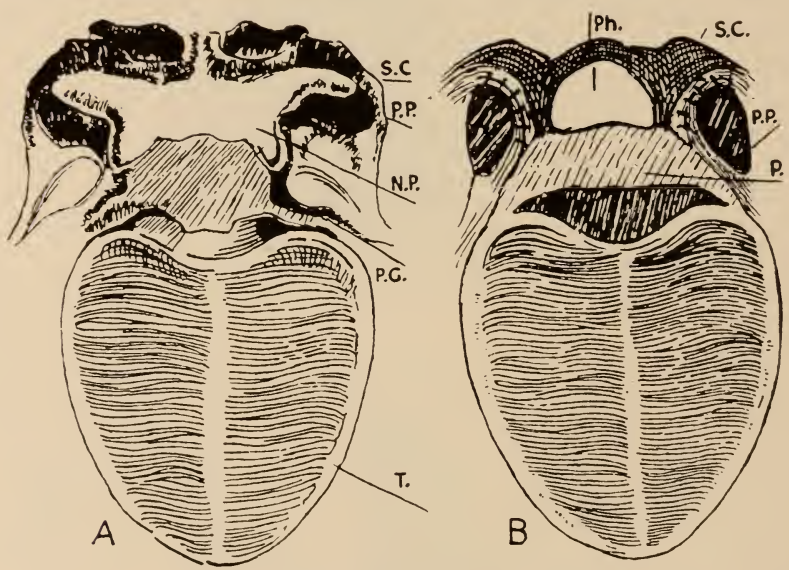


FIG. 17.—Botey's operation for narrowing the lumen of the pharynx. Note how an elliptical portion of the posterior wall is removed in *A*, and how the edges of the denuded area are joined by suture as in *B*. (From Batey.)



Cross-section of the nasopharynx just above the level of the uvula. *S.C.*, Superior constrictor; *P.P.*, palatopharyngeus; *N.P.*, nasopharynx; *P.G.*, palatoglossus; *T.*, tongue.

Showing diagrammatically a cross-section of the point where the superior constrictor was sutured to the posterior surface of the palate and the palatopharyngeus muscles. *Ph.*, Pharynx; *S.C.*, superior constrictor; *P.P.*, palatopharyngeus; *P.*, palate.

FIG. 18.—Kirkham's method of constricting the pharyngeal canal. He freshens on either side the upper portion of the superior constrictor muscle and sutures it to the corresponding freshened side of the palatopharyngeus muscle. (From Kirkham.)

2. *Operations to produce constriction of the pharyngeal canal.*

(*a*) Cauterization of the pharyngeal canal has been done by some surgeons to narrow its lumen. This type of operation may be dismissed as useless, since it produces a marked amount of scar tissue, thus disturbing the normal function of the superior constrictor muscle of the pharynx. This in turn leads to improper ventilation of the middle ear.

(*b*) Plastic operations on the pharyngeal wall are condemned, because better results can be obtained by methodical speech training which will produce a hypertrophy of the superior constrictor muscle, thus permitting the pterygopharyngeus portion of this muscle to assist the velum in securing velopharyngeal closure.

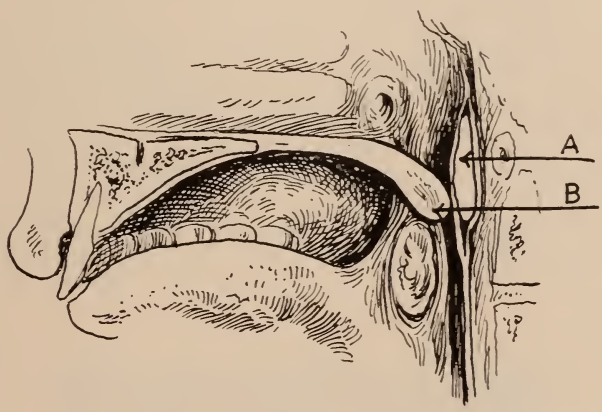
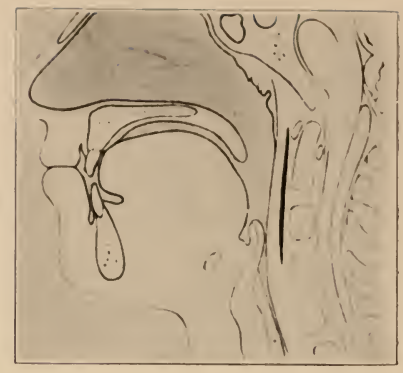


FIG. 19.—Artist's conception to show how the injection of paraffin by Gersuny and Eckstein may produce bulging of the posterior pharyngeal wall. *A*, indicates the paraffin in the retropharyngeal wall; *B*, the velum.

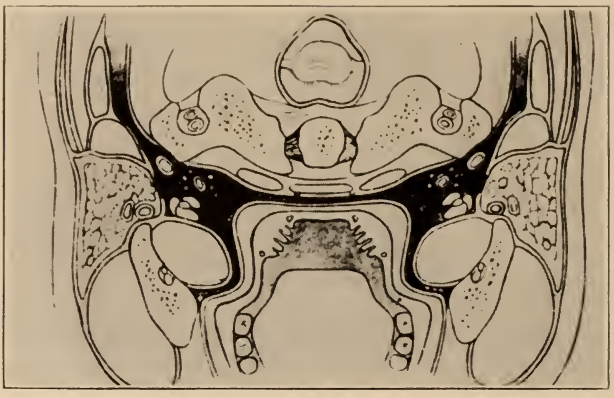
(*c*) While forward bulging of the pharyngeal wall can be obtained by the injection of paraffin or the implantation of fat and fascia into the retropharyngeal space, the first of these methods is so uncertain, as the material injected does not stay in the desired position, and the latter method (fat and fascia) is fraught with danger out of all proportion to the benefits obtained.

3. *Lengthening the velum by plastic operations* were practically all failures, since operators failed to realize two facts, viz., (1) the palatine attachment of the levator palati muscles is placed too far forward in all cases of short palate, and (2) the tensor palati muscles are shorter than normal. It does not make any difference how much

A



B



C

D

FIG. 20.—Von Gaza's operation for the implantation of fat and fascia in the retropharyngeal region. *A*, shows incision in the neck below the lobule of the ear to reach the ventral aspect of the prevertebral muscles; *B*, shows fat and fascial tissue in the retropharyngeal region; *C*, shows horizontal section to represent the extent of the fat and fascial tissue implanted; *D*, roentgenogram taken to show gold wire inserted into the implant to show it in position. (After von Gaza.)

the levator palati muscles pull, they cannot possibly bring the velum into its normal position to secure velopharyngeal closure. Attention was then directed toward lengthening the velum, by making a

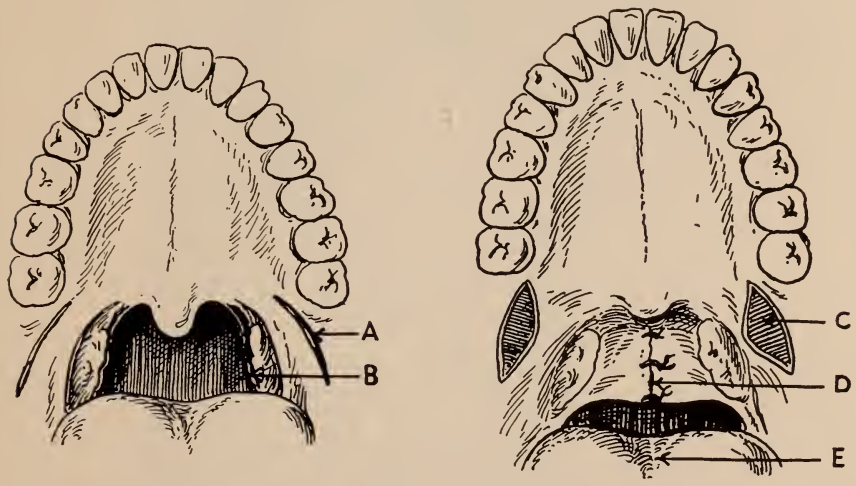


FIG. 21.—Artist's conception of Passavant's operation for suture of the palatopharyngeus muscles. *A*, indicates the lateral incision; *B*, freshened border of the posterior pillar; *C*, area to close by granulation; *D*, line of sutures; *E*, dorsum linguæ.

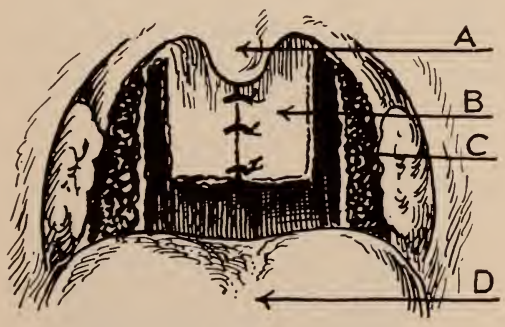
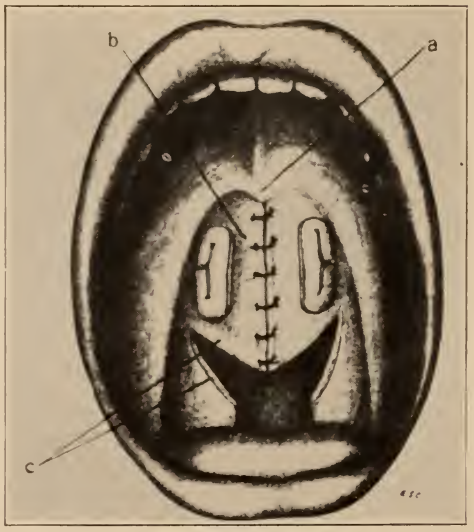


FIG. 22.—Artist's conception of Whitehead's operation, showing the use of the palatopharyngeus muscles in lengthening the velum. *A*, indicates the uvula; *B*, posterior pillars sutured in the midline; *C*, area to close by granulations; *D*, tongue.

huge uvula, which is useless in closing the nasopharynx; others produced velopharyngeal adhesion, a factor which caused many an embarrassing result.



a, end of uvula; *b*, palatopharyngeus muscle; *c*, incision made through ⅔ of width of the muscle.

FIG. 23.—To illustrate Makuen's operation for lengthening the palate by the use of the posterior pillars of the fauces, according to Brophy.

On dissecting a specimen of cleft palate, I realized the important fact that the insertion of the levator palati muscles is placed so far forward that it cannot bring the velum against the posterior pharyngeal wall, as the normally placed muscles should. Realizing this fact, it became necessary to devise some means of displacing the palate backward, so as to permit the levator palati muscles to per-

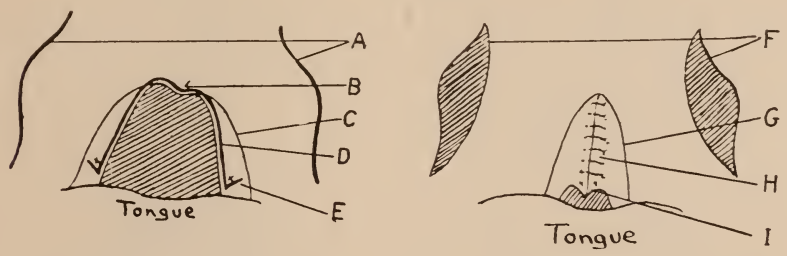


Fig. 24.—Helbing's operation to lengthen the velum, using the posterior pillars of fauces. *A*, indicates lateral incisions; *B*, "rest" of uvula; *C*, anterior pillar of fauces; *D*, posterior pillars; *E*, transverse incision into posterior pillar; *F*, areas to fill with granulations; *G*, anterior pillar; *H*, newly-formed velum; *I*, newly-formed uvula. (From Helbing.)

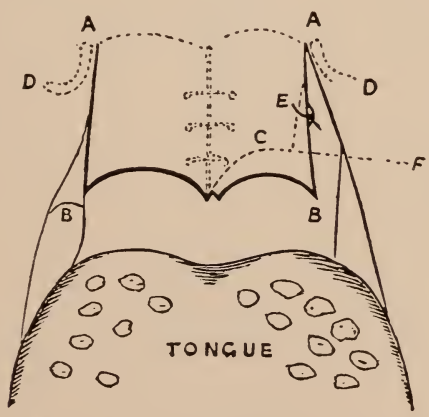


Fig. 25.—From Mason, to show his operation for lengthening the velum.

form their proper function. I was then struck by the fact that, when the dissected mucoperiosteum was completely freed from its attachment to the bony palate, the fanshaped portion of the tendon of the tensor palati, which is inserted into the palatine aponeurosis, prevented backward displacement of the flap. Division of this fanshaped portion, after freeing its attachment to the bony palate,

permitted the palate to fall backward by its own weight, so that it approximated the pharyngeal wall. This backward displacement of the velum places the levator palati muscles in a position which approaches the normal.

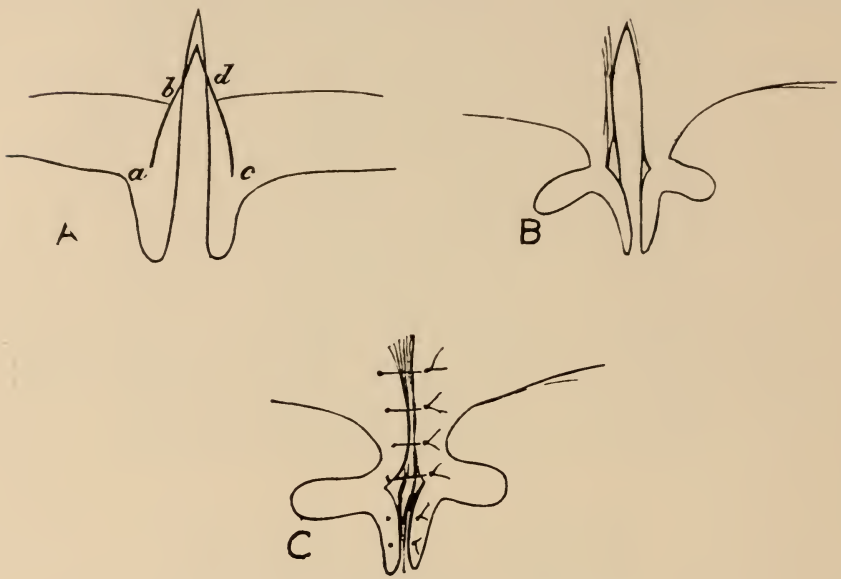


FIG. 26.—From Küster, to show his operation for lengthening the uvula.

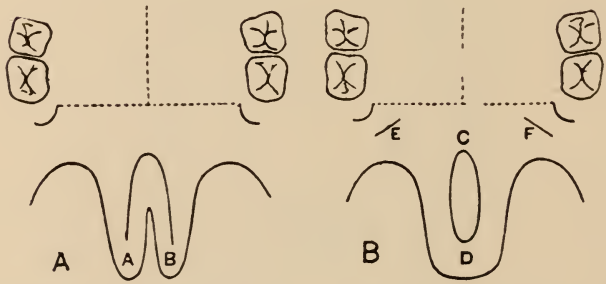


Diagram of bifid uvula. The incision is represented by the line *A B*.

Bifid uvula after operation; *C D*, oval wound left to heal by granulation. *E F*, incisions for division of palatine muscles.

FIG. 27.—Forbes operation, in which he makes use of Nélaton's harelip incision. (From Forbes.)

I then devised the following "push-back" operation.

An incision is made next to the alveolar margin and parallel with it, as outlined in (Fig. 34.)

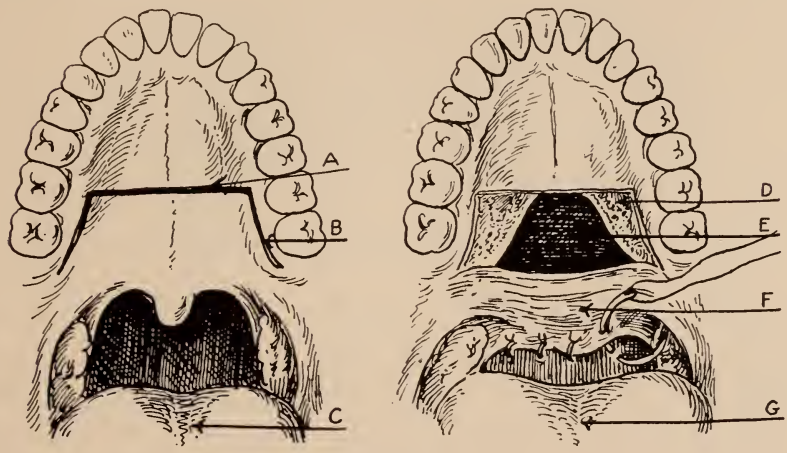


FIG. 28.—Artist's conception of Passavant's operation for backward displacement of the velum. Note how the velum is sutured to the posterior pharyngeal wall to produce closure of the nasopharynx. *A*, indicates transverse incision; *B*, lateral incision; *C*, tongue; *D*, denuded bone; *E*, nasal cavity; *F*, displaced velum; *G*, tongue.

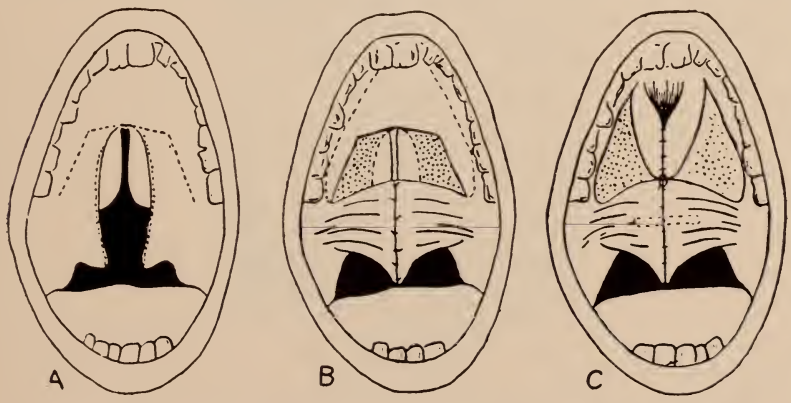
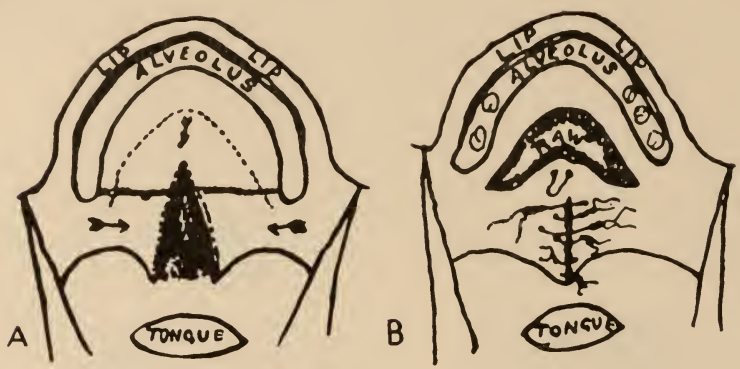


FIG. 29.—From Smith, to illustrate his operation for lengthening the velum.

This incision is extended around the tuberosity by curving the posterior extremities of the incision behind the last molar tooth. The mucoperiosteum is freed from the bony palate from before backward. (Figs. 34 and 35.)

The dissection is extended as far back as the attachment of the palatine aponeurosis at the posterior edge of the hard palate. (Fig. 36.)



A, "Apron" flap for lengthening short velum or closing clefts, using intranasal suture.
B, "Apron" flap and intranasal suture.

Nasal suture holding palate flap in position against bone, shown threaded and tied to rubber tubing at nares.

Fig. 30.—Roberts' operation to lengthen the velum by displacing it backward. (From Roberts.)

The posterior palatine arteries are divided in this operation. This constitutes the first stage of the operation. In some cases I have performed the entire operation in one sitting, where I felt the blood supply was adequate. However, as a general rule, the

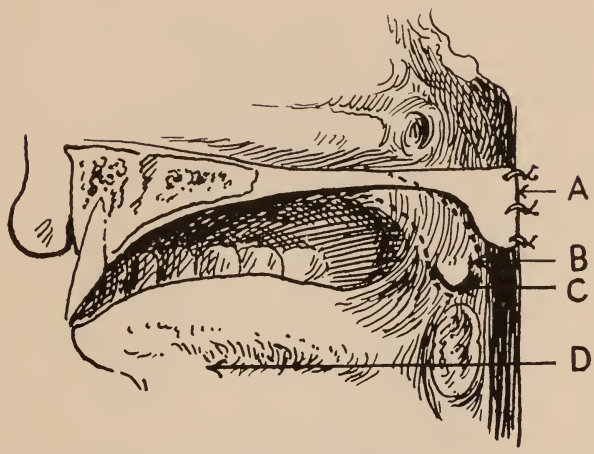


FIG. 31.—Artist's conception of Passavant's operation to produce staphylopharyngorrhaphy. *A*, indicates the velum and pharyngeal wall sutured together; *B*, dotted line shows position of velum; *C*, heavy line shows free border of velum which has been freshened; *D*, tongue.

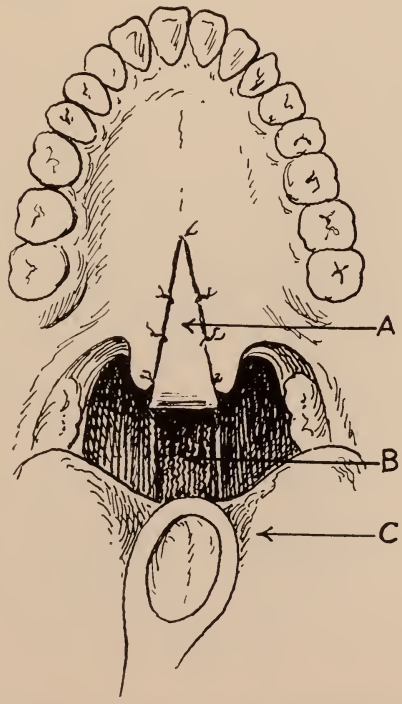


FIG. 32.—Artist's conception of Schoenborn's velopharyngoplasty. *A*, indicates pharyngeal flap trimmed triangularly; *B*, denuded area on pharyngeal wall; *C*, tongue.

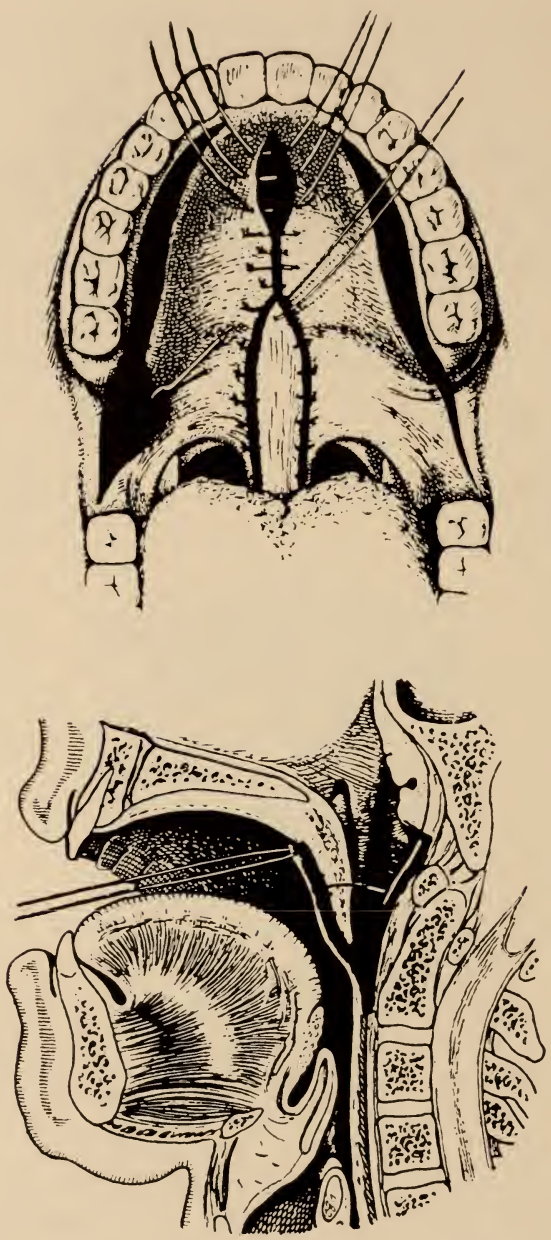


FIG. 33.—Rosenthal's modification of the Schoenborn operation to produce velopharyngoplasty. (From Rosenthal.)

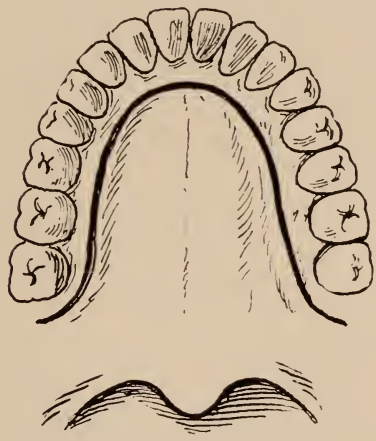
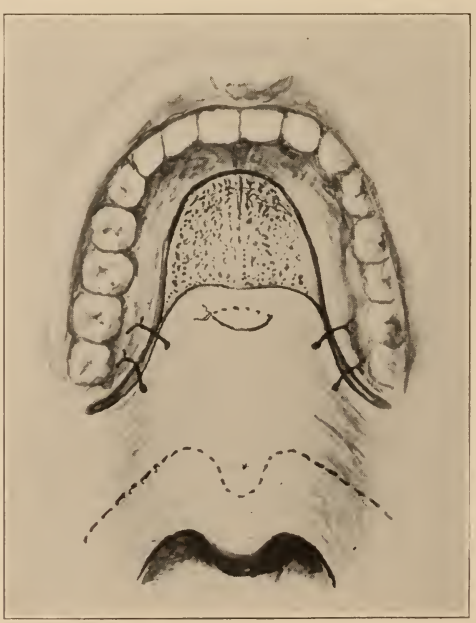


FIG. 34.—Incision to free the palatine mucoperiosteum.

FIG. 35.—Artist's presentation of case Fig. 1 with congenital shortening of palate with the mucous membrane removed.

Fig. 36.—View of case Fig. 1, after the mucoperiosteum is elevated and the palate completely freed from its attachment to the palatal processes. The fanshaped portion of the tensor palati muscle is cut.



Dotted line shows position of palate before operation.

Fig. 37.—Case of congenital shortening of the palate shown Fig. 1 after operation. Compare with original and note lengthening of velum secured.

two-stage operation is more satisfactory. The flap is returned to its original position and the edges sutured. Ten days later, when the collateral circulation is established, the sutures are removed and the flap is raised again. The attachment of the palatine aponeurosis to the bony palate is freed and that portion of the tendon of the tensor palati muscle which is inserted into this aponeurosis is also divided.

Fig. 38.—Case of congenital short palate after complete healing from the "push-back-operation."

The flap will now fall backward by its own weight against the posterior wall of the pharynx. The anterior border of this flap is sutured to the hard palate and the soft tissues on either side, as illustrated in Fig. 37.

The denuded surface remaining after backward displacement of the velum rapidly fills in with granulation tissue, (Fig. 40).

I have used this simple method with success in 3 cases of congenital insufficiency of the palate and 7 cases of shortened palate which had previously been operated upon for cleft palate either by myself

or by other surgeons, but in whom velopharyngeal closure was insufficient.

The rationale of this "push-back operation" is based upon the principle of surgery which holds elsewhere in the body, that muscles must be placed in an approximately normal position to perform their proper function. My cases have been under observation for a sufficient period of time to note the marked improvement in the functional results obtained by this proceeding.

With proper speech training, these patients can be taught to enunciate almost perfectly.

ADDITIONAL OBSERVATIONS ON THE EFFECT OF SODIUM AMYTAL ANESTHESIA UPON THE UTERUS AND ITS USE IN OBSTETRICS AND GYNECOLOGY*

By JOHN C. HIRST, B.A., M.D.

AND

MAXWELL E. LAPHAM, M.D.

Modern women do not endure labor well. New luxuries, nervous tension from participation in varied enterprises, tendency to delay the first conception, and limitation of offspring conspire to this end. There is no ideal method of relieving this suffering of childbirth. Gwathmey's rectal administration of ether in olive oil combined with alcohol and quinine and associated with morphine, with or without concentrated magnesium sulphate injection, is the most satisfactory of present labor analgesics, but has limitations. Cessation of labor pains, if used too early, asphyxia of the infant and hemorrhage from the uterus if too late in labor, short duration of relief, vomiting and other untoward manifestations may result from Gwathmey's method.

Since the first stage of labor cannot be shortened and yet must be relieved, we have undertaken the appraisal of a well-known drug, namely, sodium amytal (sodium iso-amyl-ethyl-barbiturate), from the standpoint of its usefulness in obstetrics and to some extent in gynecology. We also desired to present our report to the Fellows of the College of Physicians of Philadelphia because of finding the drug apparently useful in the management of certain medical conditions such as tetanus, epileptic convulsions, so-called puerperal mania, and prolonged hiccoughing.

We would like to say that much credit is due Drs. Raydin and Drabkin for introducing this particular work. They are responsible for the careful standardization of the drug and for the animal experimental work which was carried out.

In continuing the clinical work which we began last year with

* Read February 5, 1930.

sodium amytal (sodium iso-amyl-ethyl-barbiturate), we would like to review, briefly, the work which was covered by that report. In summarizing, we concluded:

1. In the work on the virgin guinea-pigs' uterus, either *in vitro* or *in vivo*, sodium iso-amyl-ethyl-barbiturate had no effect upon the rhythmic contractions of the uterine musculature; furthermore, the uterus of the animals anesthetized with this drug responded to the oxytoxic principle of the pituitary.

2. That the biological assay of the drug was extremely important.

3. Effective anesthesia was produced in a large proportion of obstetrical cases in which we used the drug.

4. Very little effect was noted in maternal pulse, respiration or temperature and the blood-pressure drop was a phenomenon in only 5 of 33 cases, and in these no untoward effects were noted.

5. Fetal heart-rate was very little affected and babies at delivery showed little, if any, effect of the drug.

6. We also presented the method of rectal administration which seemed to be quite effective in a large proportion of obstetrical cases.

Both intravenous and intramuscular administration of sodium iso-amyl-ethyl-barbiturate have been used in obstetrics by Zerfas, McCallum, *et al.*, with quite remarkable results and with no untoward effects, but since the fate of barbituric acid derivatives in the body is not known and, therefore, since the methods for combating overdosage are not entirely reliable and since a very careful biological assay is necessary, it would seem to us that the rectal use of the drug would be the least harmful of the three methods of administration.

The present series of 38 cases perhaps show slightly better results than the preliminary series of 33 cases. This may be due to the fact that the dosage was increased from 25 mg. to 30 mg. per kg. of body weight.

In the first 31 cases, we used sodium amytal alone in the dosage, as stated before, and obtained excellent results in 13 cases (42 per cent), wherein the patient was perfectly anesthetized and labor proceeded normally. In 10 cases (32.5 per cent) the results were listed as *good*, in which we included cases where the patients were restless with pains and required fairly careful watching. In 8 cases (26 per cent) there was extreme physical exertion, they were noisy and labor was probably slowed. It was necessary to restrain these cases.

In only 2 of the 38 cases did the patients remember anything of the labor and these were given the sodium amytal late in the second stage and the instillations were nearly all expelled. One of these retained enough to sleep between pains.

Recovery in the 38 cases averaged sixteen hours from the time of administration to complete return of mental faculties and loss of drowsiness. The extreme limits of anesthesia were fifty-two hours in one case and two hours in another from time of administration.

Thirteen of the 38 cases had a drop in blood-pressure of more than 10 mm. of mercury, but this did not last long and it rose again, except in cases where the blood-pressure was abnormally elevated by physical activity, fright or toxemia. There was no marked change in the respiration, temperature or pulse.

Twenty-three of the cases were delivered with low forceps, 19 of these with a supplementary inhalation anesthesia and 4 without the aid of anesthesia. Spontaneous delivery occurred in 12 cases, 3 of which had ether during expulsive contractions. Mid-forceps were used in 1 case. One breech was delivered without any other anesthesia, and a podalic version was done on 1 case under ether anesthesia.

In 24 cases no material change was noted in the labor or uterine contractions. In 12 cases pains came less frequently; in 2 cases labor stopped—in 1 for three hours and in 1 altogether. The latter may have been false labor. There were no severe asphyxias in the babies, only 2 requiring any degree of resuscitation; and there were no maternal or infant deaths in this series.

One of the workers suggested using some sedative in conjunction with sodium amytal to find out if we could decrease the number of poor results, *i. e.*, in the cases where the patients were violent. In a series of 5 cases morphine sulphate was used as an adjunct, given when the patient became too unmanageable. The results were not remarkable; however, this may have been due to the fact that the dosage was small—$\frac{1}{8}$ to $\frac{1}{6}$ gr.—or that it was given too late in the second stage. In 3 of the 5 cases there were no changes in the patient's activity; in the other 2 they were slightly quieter, so that we felt the morphine had some effect. Labor was not slowed by the morphine.

Two babies were asphyxiated in this series, using morphine, both of whom were delivered with low forceps under gas, the mothers

being rather unmanageable. We did not obtain the same results with this combination that the general surgeons have; however, the surgical cases do not have a long, protracted labor such as obstetric patients have. In 1 case we used 2 cc. of 50 per cent solution of magnesium sulphate, intravenously, with the sodium amytal but saw no material change in this rather unmanageable case.

We were also able to try sodium amytal in 6 cases of eclampsia which came under our care with uniformly good results:

Case I.—Primipara at seven and a half months. Had two convulsions at home; admitted and given sedative, *i. e.*, morphine sulphate, eliminative treatment, and still convulsions were not controlled. Sodium amytal was given after the fourth convulsion and the patient slept for twenty hours and had no more convulsions. Her blood-pressure fell from 170/95 to 110/90 in a few minutes and then rose to 145/90 to 150/95. After recovery from sodium amytal, the patient was treated routinely by eliminative methods and held for ten days when blood-pressure again rose to 200/110 in spite of treatment, with signs of impending eclampsia. A pelvic examination showed the cervix to be effaced and two fingers dilated, so podalic version was done under ether anesthesia, when a living, eight-months baby was delivered. Both mother and baby had uneventful convalescences.

Case II.—Multipara, eight months. Patient continued to have convulsions in spite of routine treatment. She had, in all, four convulsions; after which sodium amytal was given per rectum (30 mg. per kilo). This controlled the convulsions for eight hours. The patient did not recover enough to warrant waiting, so she was delivered two days later by Cesarean section of a living child. She was discharged in good condition.

Case III.—Multipara, eight months. History of previous eclampsia and previous Cesarean section. Had been treated for pre-eclamptic toxemia. She finally developed twitchings of extremities and face but did not become unconscious. She was given sodium amytal by rectum and slept for twelve hours. She was delivered by Cesarean section with sterilization under ether anesthesia. Living child. She made an uneventful convalescence.

Case IV.—Primipara, at term. Treated routinely for nephritic toxemia. She was given medical induction and fell into labor, after which she developed eclamptic convulsions. She was rapidly delivered with mid-forceps of a living child. Delivery was followed by two convulsions, after which she was given sodium amytal per rectum. She slept for fifteen hours and had no more convulsions. She was discharged on the fifteenth day, with a blood-pressure of 118/80. No urinary changes.

Case V.—Primipara, at term. She had pre-eclamptic toxemia and developed puerperal eclampsia. Given sodium amytal by rectum with reduction of blood-pressure, termination of convulsions and had ultimate recovery.

We thought it would be interesting to report the action of sodium amytal on certain cases other than obstetrical ones:

Case I (Courtesy of Dr. George Schwartz).—Case of advanced tetanus, marked opisthotonos, frequent convulsions. These were all uncontrolled by morphine, bromides, chloral and chloratone. Rectal sodium amytal was given, 30 mg. per kilo, and produced complete cessation of convulsions, rest and sleep, although opisthotonos continued. Patient died in spite of immediate use of serum.

Case II.—Puerperal dementia præcox, in whom wildness was relieved by one-half usual rectal dose after hyocine and morphine had failed.

Summary and Conclusion. The particular advantages of this form of anesthesia are:

1. Sodium amytal can be given early in first stage without stopping labor or without injury to mother or child.
2. It has a much longer effect than any other obstetrical analgesia without being repeated.
3. Asphyxia of the newborn is a rare occurrence.
4. Postpartum hemorrhage is not to be feared; in fact, from work now being done it would seem that sodium amytal decreases the blood coagulation-time. It was proved that this drug has no effect on the contractility of the uterine musculature and we have had no postartum hemorrhages in our two series.
5. The particular disadvantage is that there is a group of unmanageable patients who have to be restrained.
6. It is very effective in controlling the convulsions of eclampsia and it has been found to have virtue in the treatment of tetanus, mania, epilepsy and related conditions.
7. It must be carefully standardized and freshly prepared.
8. The rectal method has many advantages: (1) The intravenous administration must be extremely carefully controlled; (2) the respiratory difficulties are much more frequent in the intravenous administration of the drug; (3) delirium during the recovery has not been observed after rectal administration of the drug.

BIBLIOGRAPHY

1. Shonle, H. A. and Moment, A.: Jour. Am. Chem. Soc., 1923, xlv, 243.
2. Drabkin, D. L. and Edwards, D. J.: Am. Jour. Physiol., 1924, lxix, 177.
3. Zerfas, L. D. and McCallum, J. T. C.: Jour. Indiana State Med. Assn., 1929, xxii, 47.
4. Lundy, J. S.: Proceedings Staff of Mayo Clinic, 1929, iv, 225.
5. Drabkin, D. L., Ravdin, I. S., Hirst, J. C. and Lapham, M. E.: Am. Jour. Med. Sci., 1929, clxxviii 379.
6. Zerfas, L. D. and McCallum, J. T. C.: Am. Jour. Obst. and Gynec., 1929, xviii, 406.

DISCUSSION

Dr. I. S. Ravdin: Amytal, a barbituric acid derivative, was first synthetized by Shonle and Moment in 1923. Considerable work has been done with it since that time by numerous workers.

Amytal has certain negative characteristics which are of distinct value. Milliken and Walker have evidence that it has no effect upon kidney elimination, and the study which Bothe, Drabkin and myself are conducting leads us to believe that it has no permanent effect on body tissues, such as the liver, heart, brain, kidneys, lungs and intestines. It has no effect upon blood-pressure beyond the fact that, if the blood-pressure is elevated at the time amytal is administered, there occurs a drop of the pressure to normal limits.

These factors have combined to make many wonder whether the sodium salt of this drug would be suitable for analgesia without anesthesia. I believe we were the first to use amytal in man as an anesthetic, using it first in the fall of 1927. At that time we were somewhat concerned at using a drug concerning whose elimination we knew so little, and we inquired of the manufacturers of it as to whether or not they knew of anyone else at that time using it. Not being able to get any information on the subject after a period of time we discontinued its use as an anesthetic, although it was used at the University Hospital in a half dozen or more cases of peritonitis in which marked postoperative restlessness or delirium was present. The dosage that we used was 25 mg. per kilo.

When we became interested in its use in obstetrics we first studied what effect amytal might have on the uterus of the virgin guinea-pig. We soon found that amytal had no effect on the contractile power of that organ. It, therefore, seemed to us safe to use it cautiously in obstetrics.

Since the work was begun, the drug has been used very extensively in general surgical practice throughout the country. We have used the drug by the rectal method. The manufacturers of the drug have felt that it could be used more satisfactorily by the intravenous route, and that is the route which has been used most extensively in this country. By the intravenous route, the drug must be given very slowly—not more than 1 cc. in a minute. If it is given more rapidly than that, respiratory distress and respiratory failure may occur. Administration of large amounts of the barbiturates intravenously would seem unwise, and I think that the word of caution that was given by Soma Weiss, and which was published in the *American Journal of the Medical Sciences* last fall, was entirely proper. We have standardized each batch of sodium amytal in the laboratory before using it. When this is done, we can be assured that each preparation of sodium amytal is going to have a certain potency. The drug given intravenously must be the type of drug put out in the ampoule. Made up in that way, the drug is checked as to alkalinity and colloidal content, which is necessary when it is used for intravenous administration. Given by the rectal method, it takes anywhere from ten to twenty minutes for deep sleep.

We, however, are not thoroughly convinced that amytal should be used

as an anesthetic without reinforcement of other anesthetics. Using it as an analgesic in doses of 15 mg. per kilo by the rectal method, the patient will become sleepy in from ten to twenty minutes. The sleep is not like that observed in many of these obstetrical cases and the operative procedure can be reinforced with light gas anesthesia. The patient, after operation, sleeps for from six to sixteen hours, which is most advantageous, and the postoperative period of restlessness is not observed. In fact, in a series of surgical cases which we have not reported, we have never observed the postoperative delirium which has been so frequently reported after the intravenous administration of sodium amytal.

Dr. John O. Bower: Sleep can be induced quicker with sodium amytal when given intravenously than with any other drug. It will be interesting to know just what takes place in the cranial contents under such circumstances. We have observed the following: When a 10 per cent solution is given intravenously at the rate of 1 cc. a minute, the patient invariably yawns at the end of three and a half to four minutes. We reviewed a number of textbooks on physiology to determine the cause of the yawn, but were unsuccessful. With a needle in the intraärachnoid space, the drug was injected intravenously. At the end of three and a half to four minutes, at the time yawning took place, there was a drop in cerebrospinal pressure and it continued to drop until the patient was sound asleep. The average drop in the lateral prone position varied from 30 to 50 per cent, reaching its maximum at the end of ten minutes. It then gradually rose, regaining approximately 50 per cent of the drop at the end of fifteen minutes.

According to recent observations made by Livingston (*American Journal of the Medical Sciences*), there is an increase of cerebrospinal pressure during normal sleep. When the drug is given in solution, by rectum, the yawn is delayed, usually coming at the end of fifteen minutes; sleep at the end of thirty minutes. In the cases observed the drop in spinal pressure has not been as marked. In a fair percentage of patients sodium amytal, unless the maximum dose is given, produces restlessness and the voluntary muscles retain their tonicity. Because of the latter, the possibility of shock is considerably lessened, the peripheral circulation is maintained; therefore, it seems to be especially indicated in the weak and asthenic patient.

We have carefully studied the effect of the anesthetic on a group of 31 cardiovascular patients, to whom the anesthetic has been given forty-three times. The average age was 61.4 years; 2 were between twenty and thirty, 1 between thirty and forty, 2 between forty and fifty, 18 between fifty and sixty, 12 between seventy and eighty and 3 between eighty and ninety. All had a myocarditis: 3 mild, 6 moderate, 15 severe and 7 of a serious degree. The degree of involvement was checked by repeated electrocardiographs. Included in the group were cases of recent apoplexy, showing advanced arteriosclerosis, angina pectoris, auricular fibrillation and decompensation. The average pulse pressure in these cases was 71 (several patients had no diastolic pressure); the average systolic, 151; the diastolic, 81. The average drop in blood-pressure when the drug was given intra-

venously was: systolic, 40 (26 per cent); diastolic, 16 (20 per cent). When given rectally: systolic, 27 (12 per cent); diastolic, 15 (18.5 per cent). When given intramuscularly: systolic, 12 (8 per cent); diastolic, 4 (5 per cent). Twenty per cent of the group had a drop in pulse rate; 80 per cent had an increase. The increase in pulse rate averaged 16 when the drug was given intravenously, 8 when given rectally. Seventy-one per cent of the operations performed were major; 29 per cent minor.

From the above analysis, the rectal route, as advocated by Dr. Ravdin, is probably the safest. We have used it frequently in conjunction with local anesthesia. It is especially indicated so used, as it combats the toxic effects of the novocaine. We have used it in conjunction with spinal anesthesia in operations upon the upper abdomen, where it has a definite field of usefulness if the operation is prolonged, since the drug may be given intravenously with almost immediate effect. We have had the pleasant experience of having what would have been difficult operations converted into easy ones.

We advise against the use of sodium amytal as a preliminary, because of the change in cerebrospinal pressure, which will increase the diffusibility of the anesthetic, producing death by paralyzing the vital centers in the medulla. We have induced anesthesia by giving the drug with cocoa-butter in the form of a suppository (1.5 gm.) two hours before the operation. The patients can be aroused, and frequently complain. A few converse apparently intelligibly, but they do not remember.

I wish to show a lantern slide of a male patient, aged sixty-five years, who was admitted to the Philadelphia General Hospital with symptoms and signs of decompensation. He was markedly cyanotic, dyspneic, extremities edematous, numerous râles throughout chest, with diffuse precordial pulsations; systolic pressure, 130; diastolic could not be obtained; pulse, 120. A diagnosis of auricular fibrillation was made. This was confirmed with the electrocardiograph. Digitalis was administered, and within two weeks the fibrillation had disappeared. At the end of another week he developed gangrene of the left lower extremity. Amputation was done, using sodium amytal (rectal administration) combined with local infiltration of novocaine. The charts show no change in pulse, blood-pressure or respiration throughout the operation.

THE RELATIONSHIP BETWEEN INORGANIC DUST AND PULMONARY DISEASE*

By A. J. LANZA, M.D.
ASSISTANT MEDICAL DIRECTOR, METROPOLITAN LIFE INSURANCE COMPANY, NEW YORK CITY

The relationship between dust and pulmonary disease has been recorded by interested observers for centuries. The modern industrial scheme, however, as intensified and increased the hazards involved in this relationship, along with the increase in production.

In recent years the South African Institute for Medical Research has done an incredible amount of work on the pathological effects of dust and has made the greatest single contribution to our knowledge. England and other British Dominions have also contributed much valuable study. In this country Philadelphia took the lead, and the studies of Pancoast, Smyth, Miller and Landis and their associates, testify to an early appreciation of the importance of the subject. The first number of the *Journal of Industrial Hygiene* contained a most comprehensive article on pneumoconiosis by Dr. Landis, and Dr. Pancoast is still our court of reference in the interpretation of roentgen-ray films dealing with this condition. In fact, Dr. Pancoast and Dr. Pendergrass are the authors of the only American textbook dealing with the roentgen-ray appearance of dust fibroses.

Fields studies were and are still being made by the U. S. Public Health Service and the United States Bureau of Mines. *Public Health Bulletins* 176 and 187, dealing with the health of workers in cement plants and in the granite industry are landmarks in the field of industrial hygiene.

The effects of the inhalation of inorganic dust upon the lungs are interesting and important, not only on account of the complex nature of such effects and their serious consequences, but also because of the wholly unappreciated magnitude of their prevalence and because pulmonary dust diseases are being placed on compen-

* Read March 5, 1930.

sation schedules. The most evident effect of the inhalation of dust upon pulmonary tissue is fibrosis. Dust fibroses are classed under the general term of pneumoconiosis. Certain of these pneumoconioses are known by more specific names, such as silicosis, anthracosis, chalicosis, asbestosis, and the like, which define themselves.

Clinical evidence, all over the world, points to the inorganic dusts as being the worst offenders, and of these the most harmful is silica dust. Calcium also occurs widely through industry, as a trade dust, sometimes in combination with silica, and it is important that its effects be distinguished clinically from silica. Silica dust, in turn, falls into two classes—dust containing free silica in the form of SiO_2, and dust containing combined silica in the form of silicates. Dust containing free silica is found in hard-rock mining, granite-cutting, the manufacture of abrasives and refractories, metal-grinding, sand-blasting and numerous other industrial processes. Some of these dusts, such as result from working in quartz, flint or chert, may contain more than 90 per cent of free silica. Others, such as granite dust, range from 30 to 40 and 60 per cent free silica.

Silicate dust may contain a very small percentage of free silica and as high as 30 to 50 per cent combined silica.

Free silica dust is dangerous to life, and given sufficient exposure, the end-results are practically inevitable. The exact nature of the changes which silica causes in the pulmonary tissue and its extraordinary relationship to tuberculosis are still matters of research. When we know why a silica-dusted lung becomes tuberculous we will probably know a great deal more about tuberculosis than we do at present.

We do not have a great deal of knowledge of the effects of silicate dusts. Some of them appear to be distinctly harmful, most of them apparently will cause fibrosis after sufficient exposure, but the hazard of subsequent tuberculous infection is not clear.

In a recent article, Dr. Dublin, statistician of the Metropolitan Life Insurance Company, stated:

"Dust is next in importance after accidents as a cause of occupational disability and death. Stone dusts, which contain a very large percentage of free silica, are especially harmful and shorten the lives of men engaged in a number of industries. . . . In fact, the very highest mortality-rates and the greatest curtailment of life are found in those occupations where there is a combined liability to accident and exposure to hard stone

dust. . . . The mortality in these occupations runs over three times the average, with a resultant loss in life expectation at the age of twenty of possibly thirteen or fourteen years."

There is, unfortunately, in the United States no complete analysis of occupational mortality by which we might measure the effect of exposure to silica dust on the health and longevity of all workers exposed to it. Some facts are available, however, which indicate how serious the situation is in a few selected occupations. The most recent data are found in an investigation made by the larger life insurance companies into the occupational mortality of persons insured under Ordinary policies (policies of $1000 or more, payable at least quarterly). The study analyzes the mortality experience of those insured under policies issued during the years 1915 to 1926. The method employed was to compare the actual number of deaths, which occurred among men engaged in a specific occupation, with the expected number of deaths on the basis of death-rates prevailing among standard lives of the same ages and for the same duration of insurance. The table on page 58 shows the figures for occupations exposed to silica dust.

At the top of the list are metal miners employed underground in lead and zinc, copper, and gold and silver mines, all of whom are exposed to a serious silica hazard. The ratios of actual to expected deaths from all causes were respectively 437 per cent, 367 per cent, and 450 per cent. If we combine these three classes the ratio of actual to expected deaths from all causes is found to be 398 per cent. There were actually 44 deaths from tuberculosis among these men, where 4 or 5 might have been expected. This difference accounts for approximately 35 per cent of all the extra deaths—a far greater proportion than was accounted for by accidents. Tuberculosis was responsible for one-half of all the deaths of lead-and zinc-miners, 29 per cent of the deaths of copper-miners, and 20 per cent of the deaths among gold- and silver-miners. In all three classes, deaths from tuberculosis exceeded deaths from accidents. When we come to consider iron-miners who are but little exposed to silica dust, we find the ratio of actual to expected deaths from all causes is only 272 per cent, the excess mortality being almost entirely accounted for by deaths from accidents. There were 4 actual deaths from tuberculosis as against 1.5 expected. The ratio of actual to expected deaths among quarry-workers was 165 per cent, 7 of the deaths being due to tuberculosis were 2.7 might have been expected.

NUMBER OF DEATHS EXPECTED FROM SPECIFIED CAUSES AND NUMBER WHICH ACTUALLY OCCURRED AMONG PERSONS ENGAGED IN CERTAIN OCCUPATIONS EXPOSED TO SILICA DUST

(Ordinary Department Mortality Experience of Twelve Life Insurance Companies, 1915-1926)*

Occupation.	Cause of death.								
	All causes.			Respiratory tuberculosis.			Influenza and pneumonia.		
	Actual deaths.	Expected deaths.	Per cent actual of expected.	Actual deaths.	Expected deaths.	Per cent actual of expected.	Actual deaths.	Expected deaths.	Per cent actual of expected.
Metal mine operatives (underground):									
Gold and silver mines	44	9.77	450	9	1.12	804	14	1.81	773
Lead and zinc mines	22	5.03	437	11	0.60[1]	1833[1]	[2]	[2]	[2]
Copper mines	83	22.63	367	24	2.63	913	14	4.34	323
Iron mines	33	12.12	272	4	1.54	260	4	2.46	163
Quarry operatives	37	22.45	165	7	2.66	263	4	4.39	91
Stone-cutters:									
Granite and sandstone	29	17.47	166	16	1.64	976	4	2.87	139
Marble and limestone	12	10.55	114	3	1.02	294	2	1.75	114
Metal industries:									
Grinders	46	28.65	161	7	3.33	210	7	5.43	129
Buffers and polishers	89	64.10	139	10	6.80	147	18	11.20	161
Chippers (not ship)	38	14.14	269	8	1.30	615	17	2.27	749
Core-makers and sand-molders	32	22.65	141	3	2.92	103	9	4.70	191
Iron and steel foundries:									
Molders, founders and casters	202	130.14	155	24	13.38	179	61	22.72	268
Other operatives (drillers, flask-makers, machine hands, mixers, etc.)	45	31.03	145	7	3.03	231	14	5.19	270

[1] Approximate figure. [2] Data not available.

* Compiled from "Joint Occupation Study—1928," Actuarial Society of America and Association of Life Insurance Medical Directors.

It was impracticable to separate the granite quarriers from the limestone quarriers; were it possible to do so, it is very probable that we should find that the excess among granite quarrymen who are exposed to silica was much greater than among the limestone quarrymen. This is clearly indicated by the difference in the mortality-ratios for the granite and sandstone-cutters and the marble and limestone, the next occupations on the list. The ratio of actual to expected deaths among the former was 166, while among the latter it was but 114. There were 16 deaths from tuberculosis among the granite-cutters, against 1.6 expected; whereas there were 3 deaths in the limestone group, against 1 expected. It is interesting to observe that Hoffman, in his study of granite-cutters of Barre, Vt., found a death-rate from pulmonary tuberculosis almost ten times that of the general adult population of the state. This figure agrees fairly closely with the insurance analysis.

Grinding is another occupation in which there is exposure to silica dust, the ratio of actual to expected deaths was 161 per cent. There were 7 deaths from tuberculosis, against 3.3 expected. Here again, we have a mixed hazard, the sandstone-grinders who work in the cutlery trade have extreme exposure to silica dust and undoubtedly suffer inordinately from tuberculosis, whereas those who work on the composition wheels do not have this exposure and probably do not have very high rates from this disease. Drury, for example, found a death-rate from tuberculosis among ax-grinders and polishers, most of whom used a sandstone wheel, in a Connecticut factory of 19 per 1000. Many buffers and polishers also do grinding and are exposed to the silica dust. Their ratio of actual to expected deaths was 139 per cent. There were 10 deaths from tuberculosis against 6.8 expected.

The only other large occupation groups offering exposure to silica dust included in the study are certain selected foundry occupations. The interesting feature of the foundry operatives' mortality is that while deaths from tuberculosis were higher than expected, extreme ratios are found for influenza and pneumonia. Dust is probably an important factor also in the high nontuberculous respiratory disease rate. Chippers of metal had very high death-rates from both influenza, pneumonia and tuberculosis. There were 8 actual deaths from tuberculosis, as against 1.3 expected and 17 deaths from influenza and pneumonia, as against 2.3 expected. Core-makers and sand-molders had the expected number of deaths from

tuberculosis but there were 9 deaths from influenza and pneumonia, as against 4.7 expected. Molders, founders and casters had 24 actual deaths from tuberculosis, against 13.4 expected, while there were 61 deaths from influenza and pneumonia, as against 22.7 expected. Similarly, among the remaining semiskilled operatives (n. e. c.) there were 7 deaths from tuberculosis against 3 expected, and 14 deaths from influenza and pneumonia, against 5.2 expected.

It is evident that only dust in a very fine state of subdivision can penetrate to the pulmonary tissue—that is, reach the alveoli, and it has commonly been accepted, following experimental work at the South African Institute of Medical Research, that only particles less than 10 microns in their greatest diameter can traverse the finer air passages to reach the alveoli. The majority of particles of such dust are under 5 microns. The dust particles, having reached the alveoli are taken up by the phagocytes.[1] The phagocytes, as they become overloaded with dust, tend to block the lymphatic channels, especially the perivascular and peribronchial lymphatics. The result of the action of these cells is the production of fibrous tissue (peribronchial thickening) which spreads toward the periphery of the lung. As time passes, the fibrous tissue contracts with a consequent loss of function evidenced by a progressive dyspnea. Areas of dense fibrous tissue form in the lungs, tending to increase in size with areas of emphysematous lung between them. Pleural thickening and adhesions also form. Instead of these dense fibrous areas, hard nodules may form throughout the lung, varying in size from a millimeter to a centimeter.

The manner in which silica affects the lungs is still undetermined, although considerable research has been and is still being done. The early theory of the mechanical action of silica has been abandoned. There followed, naturally, a theory of chemical action, it being assumed that silica goes into solution in the lung tissue as colloidal silicic acid which acts as a protoplasmic poison. More recently the theory has been put forth that the action of silica is physicochemical—due to the formation of silica hydrosol—and that the various silicious dusts vary in their harmful effects in proportion to other substances present, which may tend to either stimulate or retard the formation of silica hydrosol. It is still not clear, however, why

[1] See E. L. Middleton, M.D., D.P.H., H.M. Medical Inspector of Factories: "The Present Position of Silicosis in Industry in Britain," The British Medical Journal, September 14, 1929, pp. 485–489.

silica dust produces pulmonary fibrosis much more readily and more extensively than other dusts. Not only that but we do not yet know why the silicotic lung shows such an extraordinary tendency to become tuberculous. Coal dust also causes a pulmonary fibrosis, but the victims of this type of fibrosis exhibit tuberculosis less frequently than the average. Sooner or later, in nearly all cases of true silicosis a tuberculous infection becomes activated, and there is a coalescence of involved areas with cavity formation. Tuberculosis may intervene at any stage of development of a silicosis and may be due to a fresh infection from without or to the lighting-up of a previously quiescent focus.

The silicates—dusts low in free silica, but containing large percentages of combined silica, usually in the form of calcium or magnesium silicates—exhibit their effects in a much milder form and with much less tendency toward the development of tuberculosis. There has not been nearly so much study of the effects of silicate dusts, and their pathological action is still in doubt. Some seem to produce no fibrosis; others a well-defined fibrosis, but not so disabling, nor accompanied by the characteristic symptoms of true silicosis. It is possible that the theory advanced by Heffernan[1] that the action of the silica is advanced or retarded by the chemical action of other substances present, may account for the diverse and conflicting nature of such clinical reports as are at present available.

In discussing dust fibroses, calcium dusts cannot be overlooked. Much light has been thrown upon the action of calcium dust by a recent report published by the U. S. Public Health Service[2] on respiratory disease in the cement industry.

The investigators found that there were approximately twice as many respiratory illnesses which cause disability for more than one week among cement-workers as among employed males in general, but the more serious respiratory diseases, such as tuberculosis and pneumonia, caused relatively little disability among cement-workers. The investigators state, "We were unable to find any cases of tuberculosis which appeared to have resulted from exposure to the

[1] "Some Notes on the Biophysics of Silica and the Etiology of Silicosis," Patrick Heffernan, M.D., Tuberculosis Officer, Derbyshire County Council, British Medical Journal, September 14, 1929, pp. 489–492.

[2] "The Health of Workers in Dusty Trades. I. Health of Workers in a Portland Cement Plant," Public Health Bulletin No. 176, U. S. P. H. S., Washington, D. C., 1928.

calcium dusts. Second there seemed to be no tendency for arrested cases to become active. Under exposure to the dusts generated in a Portland cement plant the progress of either active or inactive cases of pulmonary tuberculosis does not appear to be affected." The investigators did find pneumoconiosis among cement-workers, but not to an extent sufficient to cause clinical symptoms.

In trying to present to you the clinical picture of dust fibrosis of the lungs I wish to emphasize that the effects of dust exposure upon the lungs depends upon the severity of the dosage and the dosage in turn depends upon three variables. (1) The amount of free silica in the dust; (2) the atmospheric pollution by particles of dust small enough to penetrate the lung tissue and (3) the length of exposure. The larger the amount of silica in the dust and the longer the exposure the more extensive the resultant fibrosis.

With few exceptions, silicosis develops slowly. Steady work with exposure to quartz dust will produce definite evidences of lung injury in three to five years. Where the amount of free silica is less, ten to fifteen and twenty years may be required to produce a comparable amount of injury.

The most prominent, and frequently the only symptom is a progressive dyspnea which, from causing slight inconvenience may finally result in total incapacity.

These patients look well, hold their weight and feel fairly well. When the fibrosis becomes extensive they may have an unproductive cough, with a tendency to catch cold easily. Cyanosis is absent and the blood-pressure remains low. In the more advanced stages these patients will frequently complain that if they eat breakfast they will vomit it, hence they go to work on an empty stomach. Physical examination reveals little. There is diminished expansion, weakened breath sounds, and in advanced cases there may be a multiplicity of adventitious sounds, mostly of pleural origin. The dyspnea may not be evident with the patient at rest except with advanced fibrosis, but the picture of a typical advanced case with all the extraordinary muscles of respiration being used in an effort to lift the chest wall is not easily forgotten, especially when contrasted with the lack of discernible evidence for its cause. Of course, the roentgen-ray films tell the story and the physician who is unaquainted with this type of patient must become accustomed to seeing a roentgen-ray film which shows a widespread shadowing of both lungs of varying intensity but uniform distribution, unac-

companied by adequately related signs or symptoms, except a varying amount of dyspnea. The roentgen-ray shadows of silicosis begin at the hilum, and as the case progresses, extend toward the periphery, in the form of peribronchial thickening followed by small flocculent shadows evenly distributed, more pronounced at the base, with apices usually clear. The result is a very characteristic appearance of mottling. In far-advanced cases there is a tendency for these shadows to coalesce.

In the pneumoconiosis due to silica dust these shadows are harder, denser, and with less tendency to mottling than where silicate dust is the offending agent, and the roentgenologist experienced in this work can distinguish between the picture of the granite- or quartz-worker, and that due to the less harmful dusts of silicates.

Tubercle infection may occur at any stage of a silicosis. Two types of cases are seen. The first type is the usual one where there has been exposure to a highly silicious dust, such as quartz, chert, or flint. With the onset of a tuberculous infection in these cases, there comes a change, often dramatic in its suddenness. The patient loses weight rapidly, a profuse expectoration appears, there is afternoon fever, and the infection pursues its course rapidly and inevitably to a fatal termination, frequently within a year and usually within two years. Tubercle bacilli may not appear in the sputum until shortly before death. While occasional exceptions are seen, as a rule, the prognosis is hopeless. The second type of case occurs usually among those exposed to dust with a comparatively low silica content or with a milder type of fibrosis. As might be expected, these cases are milder than the first type; some do well and pursue a course similar to uncomplicated tuberculosis of the arrested or slightly active type. It may be difficult to tell whether a given case is tuberculous or not. The chief indication may be changes in the roentgen-ray film, but where the film is already fogged with an extensive fibrosis the diagnosis of a superimposed tuberculosis may be open to question, especially in the absence of signs or symptoms. Moist râles following cough are fairly definite evidence of tubercle infection. Some of the British authorities maintain that all advanced cases of fibrosis in which the roentgen-ray shadows have commenced to coalesce are tuberculous. From my own experience, which has been largely limited to the more severe types of pneumoconiosis, I am inclined to believe that active tuberculosis implanted on a case of silicosis will usually exhibit some of the signs and symptoms which we associate with tuberculosis.

Cases of mild fibrosis due to silica and silicate dusts are not detected unless search is made for them. They are free from definite symptoms, and if they have dyspnea it is not sufficient to cause them to seek medical aid. In this type of pneumoconiosis (with one or two notable exceptions) there is not the predisposition to tuberculosis which makes true silicosis so formidable a disease. These patients are occasionally picked up on routine physical examinations and not infrequently are sent to tuberculosis sanatoria because of incorrect interpretation of their roentgen-ray picture.

There is undoubtedly a great deal of this mild dust fibrosis among industrial workers. What the ultimate effects are upon the individual is still unknown. Certainly he is not benefited, and presumably he must be a more ready victim to pneumonia, bronchitis and other pulmonary infections.

The diagnosis must depend on the correlation of the patient's occupation, his symptoms and his roentgen-ray film.

Given an exposure to dust of a fibrosis-causing type, and a history of gradually increasing dyspnea, and a roentgen-ray film showing the typical symmetrical shadowing, the diagnosis is clear. We may have a typical film with no symptoms, but the absence of a typical film rules out the diagnosis of pneumoconiosis.

DISCUSSION

Dr. H. R. M. Landis: I have not a great deal to say, as Dr. Lanza has covered the subject so completely. I am quite certain that everybody here must be impressed with the high morbidity and mortality due to dust diseases. Important as this subject is, it is remarkable that it is only within the past two decades that it has attracted attention in this country. Dr. Lanza himself is one of the pioneers, especially in the use of the *x*-rays. His classification of the stages of the process, as revealed by the *x*-rays, is still used by many.

I should like to emphasize the points he has made, namely: A knowledge of the occupation, the type of dust and the length of exposure. These facts of themselves give one an insight as to the nature of the trouble and the probable extent of the damage.

It should be emphasized that organic dust has nothing to do with the production of pneumoconiosis. In industries in which the worker is exposed to organic dust and lung changes are noted on *x*-ray examination, it will be found that the dust is of a mixed character and that it is the inorganic content that is producing the damage. In a study of 50 individuals working

in the textile trades and exposed for years to organic dust, none of them at autopsy showed the slightest evidence of pneumoconiosis.

In workers exposed to inorganic dust the incidence of tuberculosis is high. If the exposure is intense and the dust consists of pure silica, tuberculosis is frequently superimposed and occurs early. In the case of potters, granite-cutters and hard-coal miners, tuberculosis occurs frequently, but is commonly a late manifestation. Since the introduction of the "jack-hammer" in the coal mines the pneumoconiosis develops rapidly—five or six years, as compared with twenty-five to thirty years under the old conditions.

Compensation for damage done to the lungs as the result of inhaling inorganic dust is coming to the front more and more. In some states compensation is granted; in many others it is totally ignored.

SAFE LIMITS OF ATMOSPHERIC DUSTINESS AND HOW THEY ARE MEASURED*

By HENRY FIELD SMYTH, M.D. Dr.P.H.
ASSISTANT PROFESSOR OF INDUSTRIAL HYGIENE, UNIVERSITY OF PENNSYLVANIA

The title of my talk implies that it is possible to determine definitely the limits of atmospheric dustiness the exceeding of which would endanger health. For toxic dust such as lead salts this is possible to a degree, and for dusts containing free silica we may estimate far less accurately with a much wider range of total amounts, dependent on several factors, such as personal susceptibility, due to the condition of the upper respiratory passages, or other unrecognized factors. For many dusts met with in industry, however, we can as yet fix no definite safe limits.

For dusts containing infectious matter, as viable tubercle bacilli or anthrax spores, any appreciable degree of dustiness is possibly dangerous, and in certain conditions the possibility of contracting these infections from dust inhalation must always be borne in mind. In tuberculosis hospitals and sanitaria every effort must be made to reduce visible dustiness to the vanishing point, and in tanneries, horse-hair dressing shops and woolen warehouses the same is even more essential. In these circumstances dust is not measured quantitatively by weight or by count of particles but potential danger is determined by bacterial culture and animal inoculation methods.

Where dusts are toxic or contain toxic or irritant particles the danger depends on the total amount of toxic or irritant material per unit volume of air. This is estimated by chemical determinations on weighed dust samples collected by passing definitely-measured volumes of air through a suitable sampling device that will entrain all suspended particles.

Such samples may be collected as solids on a cotton filter in a Buchner funnel or a glass tube, or suspended in water in an impinger sampler, to be described later, or may be deposited on the sides of the metal cylinder in an electrostatic dust sampler. In any case

* Read March 5, 1930.

the number of suspended particles is of little interest and determinations are made on the entire sample or an aliquot portion thereof.

With lead or lead salts we know that 5 mg. of lead per 10 c.m. of air if breathed daily through an eight-hour working day has caused chronic plumbism in exposed workers within a month's time. As a worker breathes about 600 cc. of air 16 times per minute or 4608 liters in eight hours, 2 mg. daily inhalation of lead has been taken as the danger limit.[1]

In tests made in a small foundry in Philadelphia recently this concentration was exceeded in all but one sample collected. Of the 30 men in this plant all but the night watchman and one other showed evidences of lead absorption and the sickness records of the plant showed an average of one worker per week suffering from actual lead poisoning. A report of these tests will be published later.

With chromate dusts met with in chrome tanning establishments and chrome salt manufacture, and other industries, we may be guided by the findings of the U. S. Public Health Service Investigation on chrome poisoning in chrome-plating, etc. Here it was determined that for safety concentrations must be reduced to under 1 mg. per 10 c.m. of air.[2]

For other toxic substances similar standards may be formulated based on known or determinable toxic doses of the substances and assuming that any material actually inhaled may be dissolved in the lungs or in the stomach or absorbed directly from mucous membranes. Drinker has shown that with a dust with particles ranging from 0.3 to 6 microns in diameter, about 55 per cent of the inhaled dust remains in the respiratory passages.

Where dusts are insoluble, or nearly so, danger is measured not by the weight of dust inhaled but by the number of dust particles ranging between rather definite size limits, and by the physical properties of the particles. Formerly with this class of dusts it was supposed that the shape of the particles was a very important factor, and we distinguished between spiculate or angular dusts and those whose particles were more or less rounded, with no points or sharp angles. Now as Dr. Lanza has told you, we recognize that the most harmful single mineral dust is silica and that it acts through it chemical as well as its physical nature. So the harmfulness of mineral dusts is measured by the number and size of the particles and also by the silica content.

As will be explained later, any standard based on the number of particles per unit volume of air must include a statement as to the method of sampling. In South Africa the Miner's Phthisis Prevention Committee has placed a standard of not over 8,500,000 particles per cubic foot (5 mg. per cu. ft. : 300 particles per cc.) based partly on conditions proved to be obtainable in the Rand mines and also on relative incidence of silicosis and tuberculosis of workers exposed to different average concentrations of dusts. These dusts average high in free silica.[3]

The U. S. Public Health Service studies in the Barre, Vermont, district state that a desirable limit would be somewhere between 10,000,000 and 20,000,000 particles per cubic foot of air for dust-making occupations for a dust which contains from 25 to 35 per cent of free silica in the form of quartz.[4]

These are the only attempts as yet to set definite standards of dustiness for relatively nontoxic inorganic dusts. Much work remains to be done along these lines. The harmfulness of different types of combined silica must be more definitely determined. English workers, until recently, were considering silicate dusts as comparatively harmless, but the latest reports state that the combination of silica dust with an alkali appears to expedite its toxic influence on the tissues.[5] Several English workers have called attention to pulmonary fibrosis from asbestos dust (a magnesium silicate)[6] and the Pennsylvania[7] and National Safety Council[8] studies indicate that silicates may produce relatively rapidly developing fibrosis.

While a number of papers have appeared in the last few years on the determination of atmospheric dustiness of various types in industry, yet those already referred to are the only ones attempting to formulate any definite standards of dustiness. As evidence accumulates it will be possible to arrive at some ideas of what are permissible concentrations for long-continued exposures. The speaker believes that even the most innocuous dust may be harmful if excessive, but at present the only accepted limits for nontoxic, noninfectious, nonsilicious dusts must be those based on the interference with vision, and the production of irritation of the eyes, nose and throat.

Various methods of sampling from dusty atmospheres have been suggested and used and as yet no universally accepted best method is recognized.

Where total amount of toxic matter is to be determined, the cotton filter, if well packed, is all that is necessary provided we have it connected with an air-pump and a flow-meter.

As dust production is rarely at a constant rate and as shifting air currents continually modify the amount of dust in a given air space, a satisfactory sampling device must sample over an appreciable time-interval to give an approximation, at least, of average conditions. The sampler must be portable, rugged and of small enough size so that it does not of itself act as a baffle and interfere with air currents. The air intake must be horizontal, or at least protected from falling particles. For industrial use the sampler must not interfere with normal working conditions, even though the intake is in the breathing zone of the worker. The rate of sampling must not be rapid enough to create an unnatural air current and draw in more dust than would be inhaled by the worker. The sampling must always be done at the same rate so that tests in different areas will be comparable.

Samples collected should be large enough to permit of both numerical and gravimetric or volumetric measurements and often of chemical analyses.

The sampling efficiency should be high, especially for particles within the danger zone, between $\frac{1}{2}$ and 5 microns for most dusts (except possibly dusts containing long spicules, as does asbestos).

There are four main types of dust-sampling devices that have been used in scientific field-work in recent years. In South Africa much excellent work has been done with the Kotze Konimeter, a syringe or force-pump device that, on the release of a spring, forcibly drives a measured volume of air against a greased glass surface, where the particles adhere and may be counted under the microscope. In this apparatus the sample volume is constant. Its efficiency is fairly high for the small sample taken, but it takes a grab sample and makes no allowance for variations in dustiness from moment to moment.[9]

The Palmer Dust Sampler used in early studies by the New York Ventilation Commission samples at the rate of 4 or 5 cubic feet a minute, the air being drawn through a bubbling fountain of spray in a specially designed pear-shaped receiver. It is portable and fairly efficient, provided not too many very fine particles are present. Palmer samples are large enough for chemical analysis or turbidity readings and are quite satisfactory for counting. However, efficiency

varies inversely with the fineness of the dust sampled, the rate of flow fluctuates and can only be measured approximately, and the sampler mounted in its case is large enough to interfere with air currents and also with manufacturing process in industrial field-work.[9]

The Electrostatic dust precipitator is probably the most efficient field dust-sampling device, but it requires more careful manipulation than do the other types of samplers. It collects a dry sample, which may be weighed dry or may be suspended in water for counting. In handling the dry sample great care must be exercised to avoid loss. The sampler acts on the principle of the Cottrell dust collector, a measured volume of air being drawn through a high tension magnetic field in a small metal cylinder. The particulate matter is deposited at the negative pole, which is the inner wall of the cylinder. Most dusts and some mists or fume can be sampled with this device.[10]

For routine field sampling, however, the most practical and efficient device in use today is the Greenberg-Smith Impinger, which is used in all studies recently made and being made by the U. S. Public Health Service. In this apparatus the air sample is drawn into a flask containing several hundred cubic centimeters of sterile distilled water, the opening of the inlet tube is either horizontal or is covered with a baffle to prevent vertical deposit of heavy particles. The bottom of the tube comes to a narrow opening at the tip, which increases the velocity of the air current at this point. It discharges vertically downward against a metal plate a few millimeters below the tip and several centimeters under the surface of the water. Suspended particles strike the metal plate at high speed and are deflected to either side and suddenly slowed up. This change of speed and direction results in wetting even the very minute particles and so entraining them in the water sample. Run at 1 cubic foot a minute this device has an efficiency of over 90 per cent for particles within the danger-size limits (0.5 to 10 microns in diameter). Where electricity is available an electric motor pulls the air through the sampler, but where no current is available a hand-pump may be used.[11]

The pump and motor may be connected with the sampler by as long a section of rubber tubing as desired and so the sampler can be suspended near the worker without interfering with his work, or a modified form of sampler may be worn strapped to the worker's

chest. For testing efficiency of dust respirators a flattened sampling tube may extend upward and open inside the respirator alongside of the worker's nostrils.

Most of the recently reported dust counts made in industry in the United States were made with this apparatus and the standards above referred to were based on impinger counts.

With any of the last three types of samplers referred to counts are made from the water suspension of dust. A measured amount of a well-mixed undiluted or diluted sample is placed in a special counting chamber and counted under the low power of the compound microscope, using a cross-ruled eye-piece, calibrated so that the rulings cover definite areas.

Formerly particles were counted and classified into four standard-size groups known as 100-unit, 25-unit, 1-unit and $\frac{1}{4}$-unit sizes, but recent reports usually neglect the larger sizes as above the danger zone and report only the $\frac{1}{4}$-unit particles or those under 10 microns in diameter.

However, the recognition of the fact that asbestos spicules up to 15 or 20 microns long or longer may reach the alveoli, will stimulate anew the recording of the larger particles, especially with spiculate dusts.

The impinger is being used now in a special investigation of the hazard of sand-blasting, being conducted jointly by the National Safety Council and the U. S. Public Health Service. Both of the speakers of the evening are members of the Committee directing this study.

The Tyndalometer is the best device for testing the absolute efficiency of dust-sampling apparatus, but is not practical for field-work, nor does it differentiate size differences in particles.[10] With this method a beam of light traverses the current of air passing through a black light-proof box and is observed at right angles through a small aperture. Even the finest floating particles are evident—as are motes floating in a sunbeam.

BIBLIOGRAPHY

1. Legge, T. M. and Goadby, K. W.: Lead Poisoning and Lead Absorption, London, Edward Arnold, and New York Longmans, Green & Co., 1912, p. 207.

2. Bloomfield, J. J. and Blum, W.: Health Hazards in Chrome Plating, U. S. Public Health Report, September 7, 1928, No. 43, p. 2330.

3. Final Report of Miner's Phthisis Prevention Committee, Union of South Africa, 1919, p. 17.

4. The Health of Workers in Dusty Trades, U. S. Public Health Bull. No. 187, 1929, p. 203.

5. Badham, C.: Notes on a Fine Type of Fibrous Pneumoconiosis Produced by Silicates and Other Minerals, Report of Director General, New South Wales, December, 1927.

6. Wood, W. B.: Pulmonary Asbestosis, Tubercle, May, 1929, p. 353.

7. Spray Painting in Pennsylvania, Department of Labor and Industry, Special Bulletin No. 16, pp. 104–109.

8. Final Report on Spray Coating, National Safety Council, 1927, p. 29.

9. Winslow, C.-E. A. and Jordan, R.: The Comparative Efficiency of the Circular Konimeter and the Palmer Water Spray Apparatus, etc., Jour. Industr. Hyg., 1923, vi, 375.

10. Drinker, P., Thompson, R. M. and Fitchet, S. M.: Atmospheric Particulate Matter. III. The Use of Electric Precipitation for Quantitative Determinations, etc., Jour. Industr. Hyg., vol. v, p. 162.

11. Greenberg, L. and Smith, G. W.: A New Instrument for Sampling Aërial Dust, Reports of Investigations, U. S. Bur. of Mines, September, 1922.

MEDICINE IN THE SUB-ARCTIC*

The Mary Scott Newbold Lecture
Lecture XXII

By SIR WILFRED GRENFELL, K.C.M.G., M.D.

It was not without considerable hesitation that I, a doctor from the wilds, accepted your kindly invitation to address you on the subject of that art to which you devote your lives at this great center of civilization and learning. I trust you will regard my effort as an illustration of the principle that I have always been taught to act upon, and have sedulously tried to hand to my own colleagues and students, that "when two ways are open, always choose the more venturesome one."

Oddly enough, the attraction, common to all challenges, is peculiarly great coming from Philadelphia. For in my student days in London, when still (though I was an Oxford student) I was unfamiliar with the location of Boston, and thought of New York only as a wild-west reflection of the gambling features of our stock exchange; Philadelphia was already in my mind as a place in which I hoped some day I might sit at the feet of the savants of my own profession. For my long-time personal friend, as well as my teacher in surgery, the late Sir Frederic Treves, was visited year after year by a surgeon from Philadelphia, the late William White. That fact alone was sufficient to assure me that Philadelphia was a place to be visited some day, for there, at least in the new world, our craft was being taken seriously.

It was at Sir Frederic Treves' suggestion that I undertook my first voyage among the deep-sea fishermen of the German Ocean, and it was after a second voyage with him that in his house in Wimpole Street I decided to leave London and the joys of a surgical practice there, to try and bring the benefits of our art into the lives of the many thousands of fishermen. These men to me embodied that genius of our Anglo-Saxon race, which through the centuries

* Read April 2, 1930.

has given English-speaking peoples their freedom, to say nothing of their place in the world. Yet these fine men were deprived by the very circumstances of their calling of the help we could render in time of need. It was so characteristic of my great chief that I want to add it was at his advice I left London in the very middle of winter for my first cruise. The boat was only a 60-ton sailing craft. The waters are the wildest anywhere. They are everywhere under 30 fathoms deep, and lie not merely "north of the roaring forties," but of the fifties also. My chief characteristically capped his advice by adding: "You will find more of interest to look at from the deck in February than in July." It was not until I questioned him about it, years after, that he added: "No man is really attracted by the challenge to wheel a perambulator on a sidewalk."

That voyage was a real voyage in the sub-Arctic, for our decks were never once clear of snow or ice. It engendered such an admiration and sympathy for the men who "do their business in great waters" that I have remained among them ever since. After all, they are the antitypes of those men whom—in spite of their ignorance, isolation and inferior social position—the wisest and most effective personality in the world's history selected two thousand years ago to "carry on" His plan for the betterment of this world.

The experiences of that first voyage also gave me a new viewpoint of etiology, and forced me to realize that germs were no more the universal cause of the troubles in the lives and sturdy bodies of my new friends, the fishermen, than any one other among the generous choice of causes given in Robert's *Text-book of Medicine*, the professional gospel on which I had been reared. For in those days, when conscious that we did not know the real cause of a disease, a large selection had to be offered.

The visits to fishermen's homes in our seaports showed that poverty was the chief factor in actual physical abnormalities, especially in the wives and children. Even in those days it was easy to diagnose their troubles as "deficiency diseases," though yet we knew nothing of vitamins. Roberts had never mentioned poverty, much less the selfishness and sin that caused it, in his otherwise generous lists. This discovery was, for us, a greater epoch-making event, in enabling us to do effective preventive medicine for thousands of fishermen, than was the laboratory discovery of insulin, or even Ehrlich's "606."

One cannot prevent poverty with vaccines, or destroy it with

antiseptics. Indeed, alcohol was itself the cause of most of the trouble, and that not merely by cirrhosing organs and hardening arteries, or even by causing interesting surgical cases, which are politely attributed to "criminal instincts" or "accident," and which now so many are attributing to the "spirit of independence" exhibited in resisting the relegation of that particular toxin to the drug cupboard.

As a matter of fact, our people in the North Sea earned excellent wages, but the brewers and distillers alone got rich on them. In the seaport in which I made my first headquarters, I published the list of over 100 open saloons along the wharves which led from the spot where the fishing boats coming from sea tied up, and the homes of the men. Vessels, loaded with alcohol, pursued the fishing fleets on the high seas, where resulting ataxia was not infrequently the cause of very sudden death. Yet "scientific" textbooks have not even yet suggested that some of my wealthy friends, some even ennobled for their activities, might well have been added to the list of causal factors of death and disease.

Intelligent patients are generally interested in the cause of their troubles. My patients were, and they only fell as a result of temptation, after endless struggling, exactly as the morphine victim does.

I have spent so long on this one "unscientific" aspect of the challenge to the country doctor, because it symbolizes many less obvious apologies for his existence. Fortunately for us, it had never paid the owners of fishing vessels to have their crews at sea drinking alcohol, as they often sold their ship's gear for it. So without any taint of sentimentalism, they, together with the insurance companies and taxpayers, helped us procure international prohibition of the sale of this inessential toxin to fishermen on the high seas, and no one, not even the rich, has ever seen any reason since to repeal the act. So law enforcement was easy.

It was in 1892 that we extended our sphere of action from the North Sea to the coasts of Labrador and North Newfoundland. This new field presented problems in an entirely fresh way. For while again the "floaters," who fished entirely from their vessels, had practically no access whatever to professional help, there proved to be a large scattered English-speaking population who fished only from the land; in which no resident doctor, trained nurse or dispensary had ever been known. After our first cruise of three months in a small schooner, without power, the freezing sea drove

us out, and I found myself sailing back across the Atlantic, studying my records of over 900 cases, which included almost every kind of surgical need, and arrears of medical neglect that were challenge enough for any one man's life anywhere.

Some center for collecting the sick was a primary necessity. One had been taught to believe that a hospital in the hands of a rural general practitioner was essentially a shambles. Consultant friends in Harley Street had often regaled me in old days with stories-that-may-not-be-told of the delinquencies of the rural practitioner, and which, alas! were doubtless only too true. Indeed, the best known of all my surgical friends once had come to the conclusion that "while medicine offered the best education in the world, its subtle temptations made it the worst profession out of which to earn a living."

On sailing back for England that first autumn, however, I had ordered the Labrador settlers to cut the frame for a hospital. A hospital under such circumstance had no less attractions because it had to be a sporting venture. Two well-qualified young doctors and two of my old, highly trained nurses from the London Hospital came out with me the following spring and, shortly after, a second hospital 250 miles north was started on an island called "Rodney Monday" in Indian Harbor, directly in the track of the fishing fleets.

The actual professional work of these first two seasons had left us absolutely determined to return, so while the others sailed back to England, with my colleague from Battle Harbor Hospital we went to Newfoundland and Canada to try and enlist sympathy and money. Newfoundland received us more than graciously, and promised a small Government grant, and free entry of all supplies (far the most valuable concession), one merchant donating a building site for a hospital.

Unquestionably, it is better that in a city practice the doctor should be spared the burden of raising the budget. With us this was impossible, and I am convinced, much as I hated the task, that we received a better hearing because of the profession of which we were members.

As a result of our winter's work, the following spring I steamed a new small hospital steamer from Montreal to Labrador, visiting as I went all along the North Shore of the Gulf of St. Lawrence, and expecting a triumphant arrival at Battle Hospital, where not only my colleagues but the schooner from England was awaiting me

with supplies. Three miles from hospital, however, between two islands, after 900 miles of safe navigation, we ran full speed onto a reef of rocks, where besides stranding we incidentally lost our shaft and propeller. Hours later, all alone and unannounced, an ashamed and heart-broken general practitioner climbed down the steep cliffs into Battle Harbor, desiring only a lethal dose!

There was only one chance to save the new boat. We must at once float her, and the sailing schooner, very little larger than herself, must try and tow her across 350 miles of open Atlantic Ocean for repairs. This was safely accomplished, though it took all summer. Meanwhile I had to wander along the coast with a small portable outfit, getting passages as best I could.

Time appeared to be wasted, work to be inefficiently done. Many opportunities had to be neglected; and *yet*, with that utter lack of logic that seems to characterize human affairs, when winter came we had somehow got actually nearer the hearts, and real selves, of our people and consequently of our problems than we could have possibly done in any other way. Essential as it is that our profession should meticulously guard its scientific basis, and its impeccable ethical methods, it cannot afford to forget that the world is run on emotions, and not on logic. A sympathy engendered by experience had sunk into my soul and theirs, which has served to encourage us to "carry on" when every one was urging us to give up.

Per contra, it must not be forgotten that wherever any one interferes with entrenched conventions, opposition is inevitable. Thus, on one occasion, walking over an island to see a patient, I stumbled over a girl of about eighteen years, lying drunk by the pathway, not far from a big trader's house where liquor was freely sold. What good was advice? What kind of drug or treatment could remedy the damage done? That trader and I parted a little later, he threatening to have his men throw me in the sea if I ever landed again on his island, I assuring him I should at least not quit until I succeeded in prophylactic measures which would include removal of the causes of cruel suffering.

The relative importance of mental agony was impressed upon me in another way. I had once been called by a half-masted flag to a fishing schooner, while cruising with a fleet near the entrance to Hudson's Bay. Going aboard, I had found a lovely girl of eighteen lying in a bunk in the open cabin, bloodless and collapsed, in blankets soaked in blood, among which still lay a dead baby, exactly as

it had been born. Wrapped in a couple of blankets, we lowered her over the side, carried her aboard my hospital boat, and steamed at once for a little Moravian Mission station at Ramah, where we left her under the care of the good housewife. In the fall we stopped to pick her up, for no mail-steamers went so far north. Until we had started south, she had not suggested remaining on the coast, even had that been possible. To my amazement, on the first day out at sea, she insisted she could never live to return home. Pulse, temperature, examination revealed nothing abnormal. Yet she faded out in a few days. We just wrapped her in her country's flag and buried her body on a rocky headland, where a rude cross still convinces me that the mind is a factor in treatment which the laboratory cannot afford to discount.

To explain further how unavoidable is opposition, except to a clam in the mud who does nothing, I should say here that the co-operative stores, the interference with liquor customs, the hospitals and their implication of professional neglect, the schools, and especially the necessity for reports and lectures which the budget involved, indeed, any and every reflection on privilege and custom, even the mere absence of any particular ecclesiastical affiliation, inevitably breed criticism, resentment, opposition. We have all seen this operate, even on such exalted servants of the public as the President of this great nation.

It was not until two years later, when the saloonkeeper, who had possibly forgotten the incident of the girl lying drunk by his roadside, gave me my chance to make my prophylactic efforts effective in his case.

That fall my boat, always "the keel of the Labrador," had gone into winter quarters. My crew was paid off, and I was just leaving St. John's when the morning paper announced the loss of a large foreign schooner loaded with a cargo of fish for the Mediterranean, 600 miles north, near the entrance of that harbor. The unusual lateness for a vessel to have lingered there attracted my attention. The same day I hired a steam trawler, shipped a diver and left for Labrador without telling any one. The quest was as exciting a venture as that of Robert Louis Stevenson's "wrecker." We found that wreck, butted our way in through ice as close to her as we dared, and put our diver underneath. Finding that the protest we had read at the St. John's shipping office was obviously false, we jettisoned the cargo from the wreck aboard our vessel, floated her,

rigged her by raiding the Labrador storehouses of our friend, the saloonkeeper, put one-half our crew aboard her and towed her some 600 miles south; finally anchoring her in the middle of St. John's harbor, dressed in all the flags of both ships, from her bowsprit end to her jigger boom. The result was that the saloon was closed, never to reopen, while our friend was more worthily employed in his Majesty's penitentiary, making brooms.

Oddly enough, centers for selling supplies as therapeutic efforts became later essential in Labrador. It was stipulated on the transfer of the site for our first hospital that we were never to sell pork or molasses from it, as the universal truck system of trade which prevailed gave that monopoly to the traders. But again, the resultant poverty forced our hands and led to the establishment of a series of cash coöperative stores, which helped both to abolish the truck system and to put such luxuries as milk, sugar and more ample food supplies in reach of our patients, for whom before it was useless to prescribe them, unless we provided them ourselves, free.

The starting of the coöperative cash stores, however, brought us more serious opposition, and we woke one day to learn that at the request of many merchants, all of whose names were recorded, a commission was to be sent down by the Government to investigate our irregular incursions into commerce. The list of accusations, however, were not confined to coöperative stores. Every deparment of the work was under attack, and even (we being protestants) our doctors were accused of sectarian discrimination. This was made possible by intense sectarian fears, arising from the fact that all the educational grants in the country are divided "per caput" amongst the leaders of the varying sects and creeds. I have thought it appropriate that this particular tale should be retold in Philadelphia, for when the commission sat in our headquarters at St. Anthony, the medical officer in charge of the station was from this very University of Pennsylvania. Perhaps not unnaturally, the procedure on that occasion is fixed in my memory with unusual distinctness, for I subsequently printed 20,000 copies of the whole findings, for free distribution.

"Are you a doctor of medicine?"

"Yes."

"What is your name?"

"Louis Fallon."

"Your university?"

"Pennsylvania."

"Are you in full charge of this station?"

"Yes."

"Do you discriminate in your treatment of patients?"

"No."

"Do you give a Roman Catholic the same treatment as a Protestant?"

"Yes."

"What is your salary?"

"I am a volunteer, without salary."

"How long have you been here?"

"Six years."

"What is your religious affiliation?"

"I am a Roman Catholic!"

The reports were criticized as the best publicity that this work could possibly have received. But the mud had been slung.

One must pass over many other prophylactic efforts that as empirical practices amply justified themselves as "scientific," even if the actual ventures were originally based on spiritual rather than on physical premises. Fishermen have ever been men of faith and courage, and the popularity of the dispensary lay in the fact that they have the greatest confidence in anything in the form of medicine. On one occasion, I happened to visit a vessel after having sent aboard a bottle of medicine. To reach my patient in the bunk, I kicked a big sea-boot off the settle, when out fell my bottle and broke on the floor. On my upbraiding my patient for carelessness, he excused himself by saying he had hidden the bottle there.

"You never told me any of the others were ill," I countered.

"None of them are," he replied. "But they would drink it if they found it, fearing they might be."

That partly accounts for the weariness of long clinics on the Labrador Coast. For the fact is that not only do the sick come, but those who have been sick since you saw them six months before love nothing better than to be "sounded," and told "All A1 at Lloyds," while the rest, feeling sure they will fall sick before they see you again, are equally eager to get your advice as to how to act if they do. In Labrador, it had been the custom of the Government mail steamer to hand out patent medicines to fishermen seeking aid. I have known mothers in Labrador, who for years never missed

that opportunity, and who possessed enough infallible remedies to cure all the Coast of anything. The value of faith as a healing factor is often unusually illustrated on the Labrador. On one occasion, a great powerful Irishman came aboard, his head all tied up and himself in great distress. He had an abscess beneath his third molar, and a partial trismus, so that he could not eat. There being a better light on deck, I told him to wait while I fetched the instruments. Alas! being nickel-plated they flashed in the sunshine, and mistaking them for knives, he would not let me near him. It ended in his insisting on being charmed from a distance, which he said I would do for him were he a Protestant. I muttered some Welsh. He paid the fee, and a fortnight later when I saw him again, he swore he had never had another pain after this charm was uttered.

The greatest lesson a country doctor has to learn is patience. With us, the distances are so great the temptation to haste is as resistless as in New York, or Philadelphia.

It is with minor ailments, or what appear as such, one finds most difficulties. When a man is brought aboard with both arms shot off, or drags a limb that has been useless ever since an accident of ten years previous, and proves to have been just "out of joint," one is naturally interested in the problem. But when two dozen big brawny fellows have been aboard in one day, all complaining of "bad backs," and you cannot feel or see one thing wrong, you are apt to suspect and discount even the implicit faith that brings the patient to your consulting room, more especially as the fee of a few cents, or some fish or other offering to the ship, seems an inadequate return for your outlay.

I had to make several visists to a famous Boston orthopedic clinic to rekindle my interest in backs, until once, when hauling an anchor in rough water, I got one myself. From that time sympathy with any one who has to go on hauling nets and ropes and anchors hour after hour, day after day, has made even bad backs almost a hobby, without committing myself to osteopathy or anything but the most conventional of efforts. Moreover, fortunately for Labrador, it enabled us later to induce Dr. Goldthwait, of Boston, to visit all our hospitals, which he most generously did, purchasing a fine 15-knot steamer to save time, and eventually capping his free services to the Coast by presenting us with the boat as well.

Among other activities, we have had to regard many additional

expenses, such as transportation, as parts of our "medical" problem. This had to be by sea in summer, by dogs and sledges in winter. Hospitals cannot exist if patients cannot get to-and-fro. In maternity abnormalities we try to map all such troubles as rickety or contracted pelvis, and send them in good time to places where we can get at them. Even then we are at times too late.

I was called one winter to a maternity case in a lighthouse, 30 miles distant. My colleague, Dr. Louis Fallon, of this university, answered it. Heavy weather, deep snow, and bad salt-water ice held him up, and when he at last arrived the trouble was over. An abortion had occurred and the mother seemed easy and in fairly good condition. The dejecta preserved for the doctor to examine proved of great interest, and Dr. Fallon brought it back with him when he returned to the hospital. It proved to be a complete uterus, including the upper end of the vagina, both tubes and one ovary. The uterus itself, if I remember right, was about 4 inches long. The specimen is now in the Harvard Museum. It is the only spontaneous panhysterectomy I have ever seen, and was particularly interesting, Nature had performed, by a sharply-defined ulceration, the operation for which, when it was first introduced into Boston, the late Dr. Storer of Newport, told me nearly cost his expulsion from the Medical Society. The cause of the trouble, under the circumstances, was never discovered, but the woman was not subsequently troubled, except by the loss.

Better facilities by mail-steamer, and the invention of motor engines, have been able to modify somewhat the purely medical work of my hospital steamer to the task of a liaison boat, and a social activities director.

Many cases a hundred or more miles distant from the hospital often needed immediate aid, while such a large proportion of cases were due to deficient or imperfect diet that much child welfare work was imperative if we were to benefit the scattered villages and did not wish to measure the importance of our work merely by the list of major operations.

Coöperative distribution was accomplishing something, but more production to give more remunerative work in off seasons was essential. For this purpose, a lady from Providence, Rhode Island, a master of weaving and other arts, came north at her own expense and for many years not only fostered the hooked rug and other industries of the forebears of our people in Scotland and England,

but also taught many other new ones. Gradually a regular industrial department developed. This was soon associated with occupational therapy work, for the psychology of a fisherman lying sick in bed week after week, conscious that he was earning nothing, and that his family was growing hungrier and nakeder, and his means of earning was rapidly deteriorating, demonstrated clearly the importance of the psychic factor. The doctor, who was watching the long process of fusing a spine or hip, could not but look with gloomy foreboding at the prospect of his patient's return to immediate want, or to physical labor of the hardest kind; either of which might easily undo all the value of his efforts. Today, a specific attempt is always made to rehabilitate each patient as a part of the scientific challenge to the doctor. Thus, for example, last spring, a father of a family, whose leg had been removed two years previously for tubercular disintegration of the ankle, came to hospital with white swelling of his wrist. Six months' fixation and hospital care during the summer relieved the local condition, but as he had a wife and nine children to return to, and as he had lost his whole season's fishing, it would have been kinder to have lethalized him at the beginning than to send him home. As it was, he returned able to carve, with a complete outfit of tools, material and orders to keep him busy all winter—an actually scientifically cheaper cash outlay than to have him back in hospital for another long period next spring, from recurrence.

The industrial department began with an output that was the ridicule even of our own harbor. It has had to grow without capital and at the expense of much anxiety to the directors of our general fund, to which today it owes a very large sum of money. But we have a shop here, at 1631 Locust Street, and one at 425 Madison Avenue, New York, selling on their merits beautiful rugs, weaving, carvings in ivory and wood, etc., as an odd outcome of general practice in Labrador. The turnover now exceeds $50,000 per year, and helps over 2000 people. Thus, we actually sell on the Coast, or here, almost everything, as in some way part of a country practitioner's technique.

Our schools—one the gift of Yale boys, another that of Princeton boys—are yet unable to afford supplementary technical training. Such numbers of scholars as we can afford to finance each year are in training in such splendid schools in this country as the Pratt in Brooklyn, Wentworth in Boston, Berea College in Kentucky, and

in agricultural, business and other schools in Canada or the United States, and even in England, where scholarships can be obtained. We have developed in the course of "general practice" our own machine shop, our own dock and repairing shop, and a tannery for enhancing the value of our seal catch, a small lumber mill, and several centers for farming and for improving by skilled adaptation and supervision the economic output from both the flora and fauna of the country. We have been able to show the Coast could supply all the vitamins needed to prevent disease, that once took such a terrible toll of our people.

A child welfare department, started and maintained by college girls, has introduced goats and better hygiene, and much improvement and economy in the purchase and preparation of foodstuffs. In the early days of high prices of adrenalin and pituitrin, we secured an industrial chemist from Kansas, Dr. E. B. Weidlein, now President of the Mellon Institute at Pittsburgh. For the adrenal body of a whale was 2 feet long, and the pituitary gland as large as a man's fist. The synthetic article, however, ruined this hope for an economic asset to the Coast.

Our last hospital was built entirely by Labrador boys trained under our care. It is fireproof, central-heated, electrically lit and with all the amenities of running water and good plumbing right through the Arctic winter. It has also most of the modern hospital special equipments, and has been standardized by the College of Surgeons of America.

The care of derelict children has had to be added to our opportunities. There is still no provision for the underprivileged child in the North, except what we can offer, or for the deficient-minded child, the moron, the blind, the hopelessly crippled, or the children left without any bread-winner or natural protector (unfortunately not an uncommon occurrence in a life such as our men live). For example, just before this very Christmas, close to my own hospital, 5 bread-winners returning with the Yule logs by motor boat, were swept out to sea by a sudden gale, and never heard of again.

Another reply to the problem of our widespread parish has been the establishment of small nursing units, half-way between hospitals, under efficient nurses, with public health and maternity training. The buildings contained 2 or 3 spare beds, a small dispensary, and after the manner of the original welfare workers (who worked best in pairs), a trained social and industrial worker to act

as comrade and assistant to the nurse. It is remarkable to note the excellent medical results which these workers have accomplished. Professionally they have attempted almost everything; for instance, Nurse Jardine, of the Massachusetts General Hospital, with complete success, replaced viscera, cleansed and sutured an abdomen that had been ripped open in delirium; while Nurse Bailey, of London, operated on and saved a horribly wounded shoulder-joint caused by the discharge of a gun through the axilla. These nurses also carry on dental clinics, supervised by our dental surgeons when they make their rounds. Of these, Harvard has sent us no less than fifty.

When visiting specialists have come along, such as Dr. Goldthwait, Dr. Rugh, Dr. Lockwood of Pasadena, Dr. Andrews of Santa Barbara, or Dr. Curtis Eves, who is here tonight, and others, these nurses make special clinics possible locally, even if the beach had to be commandeered for an operating theater. This I remember was the case at Forteau Bay station, when I chanced in there in the hospital boat and found Dr. Eves at work!

The Government aid in all these matters has been confined to giving free transportation for patients, free duties to the work and workers, and a small, though welcome, subsidy to the hospital budgets.

The recurrent "dry-face diet," indicating a minimum of white flour, raw tea and nothing else, all of which we saw every spring, before supplies for the year could again reach the villages, left no loophole by which we could avoid mixing up in struggles which interfered with other time-honored conventions. Rickets, acute and chronic, were in every village. Ill-nourished mothers, unable to suckle their offspring, and equally unable to obtain the "luxury" of canned milks, had learned by experiment and necessity to predigest flour in their own mouths, in order to carry a scrawny baby through until its salivary glands developed. We found endless purpuric complications which confused diagnoses, foiled attempts to cure, and even led to death from true scorbutus, and yet in the spruces and endless wild berries, easily preserved by freezing, the remedy lay all around the people.

Undiagnosable by us personally, at first, was one trouble generally beginning with excruciating pains in the legs, and going on, if untreated, to paralysis and death. It turned out to be beri-beri. This disappeared, as a rule, after hospital treatment, owing to

improved diet. Usually it attacked the fishermen after the long winters, just when their fishing season was commencing, and by preventing them earning what is called locally "next winter's diet" created quite unnecessarily a very vicious circle.

The clothing problem was tackled by a regular system, supplies coming from England, Canada, or the Needlework Guild of America. Work was always exacted in return where possible, lest the remedy should be worse than the disease, and this was made possible by the Industrial Department. Thus, besides lack of knowledge, poverty was, as in the North Sea, an unquestionable etiological factor in a large group of troubles, and to fight it became a rational part of the doctor's task, exactly as attacking the "unseen world around us" with antiseptics and instruments is becoming everywhere recognized as "religious" propaganda.

Law enforcement is far from a new American problem. There being no regular magistrates for the North, we accepted also that most ungrateful task of unpaid and amateur dispensers of justice, to which we could bring only a sincere desire for equity without knowledge of precedent. It appeared to work satisfactorily, however, so long as no specialists of the sister profession of law were present to confuse issues, and intimidate our courts. Moreover, we were permitted to make our sentences "secundem artem," remedial rather than retributive, and for lack of other entertainments our court trials were always exceedingly popular occasions.

The absence from my records, and a general audience, modify the value of more purely professional deductions, but I feel that some will still be of interest.

Leprosy. In London I had become familiar with the signs and symptoms of tubercular leprosy at the London Hospital, where Sir Jonathan Hutchinson was teaching that it was due, or largely due, to eating dried fish. In forty years in this fish-eating country not a single case has been seen.

Tuberculosis. Our people live an almost entirely open-air life. On the other hand, the cabins and holds of fishing vessels on which many live from four to six months a year are almost hermetically sealed, are dark and almost always damp, and like the cottages ashore are all small and almost impossible to keep warm and ventilate at the same time. The larger cottages, built of board and wooden frame, are cold, and impossible to keep free of draughts. The majority, however, are small log and mud-covered huts, dark

for want of glass, at best. Windows generally do not open, and in summer are kept closed also through the hottest weather, as no attempt is made at screening, for want of material. Many pulmonary cases we have kept under shacks of old sails, just as Sir Robert Jones began his famous orthopedic hospitals under sheets of galvanized iron in the middle of the open fields.

Tubercular diseases of all the tissues are still very common, and tuberculosis still takes far the largest toll of our people. Now, every year, we carry down mosquito netting for the people. There were then no cows in the country, and few were able to purchase preserved milk, or get it on credit. Thus, when patients came to hospital it was almost impossible to induce them to drink milk. Most would not do so. Some could not drink it. Yet bone tuberculosis was as common as pulmonary, and still is so. Mothers, often through malnutrition and deficient diets, often could not nurse their babies. They, however, knew their babies could not live on "loaf," or soaked hard bread, which they called "pap." I have often seen mothers chewing the starch food themselves and then feeding their offspring. They had learned how to make it digestible, but of course had no idea of the reason.

Syphilis and Gonorrhea. Venereal diseases are rare among the residents of the Labrador Coast, or the Newfoundland fishermen, and there was none at first that we could hear of among the native Eskimos, among whom the Moravians have worked for one hundred and fifty years. In 1892, some Americans took a schooner load of Eskimos to the World's Fair at Chicago. Those that returned brought back both infections. The following winter, owing to the inherited habit of more or less freedom of intercourse, possibly necessary among the northern Eskimo women in order, under their hard environment to maintain their population, almost every woman (and I examined almost all) was infected, anywhere in the neighborhood of Hopedale. We used mercury and chalk in those days, and we had to send a barrel full "down North" after returning that fall, every atom of any syphilitic drugs in our dispensaries having been exhausted. The mortality was great, and incidentally, a census taken a few years later showed that there was not an Eskimo mother on the coast who had given birth to 3 children that had not lost 1 before one year of age, and the 3000 Eskimos in Labrador, in 1892, have now dwindled to less than 900, and that includes a fresh influx from Hudson's Bay and the North.

Another point about these diseases was the menace of the fact that in our territorial waters, American fishermen had retained the ancient right of pre-Revolution days, to fish within the 3-mile limit, and to use the harbors and bait grounds. Our people are simple and deeply religious, and (whether "propter" or only "post hoc") were like the Eskimos, also, singularly free from venereal diseases, unless banking vessels from southern ports came in and used our harbors. Then the glamour of these wealthier visitors often spelt disaster to us. As Magistrate as well as Doctor for the Coast, I have had, for lack of policemen or jails, to order a vessel at the muzzle of our ancient fowling pieces to leave the harbor, as they carried infected members of the crew.

Malaria. No country I have visited is more beset by the mosquito in the summer months. They and the black fly make it impossible to fish even the most alluring and virgin rivers, without elaborate precautions for protection. Yet we never had a single cause of malaria or yellow fever. I may add we know of no endemic or indigenous diseases. I can only personally remember 2 cases of liver abscesses, and both were sailors on foreign-going vessels.

Dysentery. In spite of the utter lack of sanitation, drains, or any of the usual health precautions, we have very rarely any specific intestinal affections. I can call to mind only 1 case of amebic dysentery, and that was in a visiting clergyman, who eventually died of it in hospital.

Tapeworm. Various intestinal parasites prevail, and oxyuris and ascaris are very common. Every house owns half a dozen wolfish Eskimo or mongrel dogs, for transportation purposes. The dogs live always around the houses, and in them, and often no attempt is made to clean up the intolerable messes they make. Yet we have never seen a single case of hydatids. The famous Dr. Cook was good enough to bring me over a small consignment of husky dogs from Greenland, but fearing possible infections from the echinococcus, we felt forced to shoot them.

Specific Fevers and Immunity. The approach of specific fevers is generally heralded in advance. We have several times had measles, scarlet fever, and even smallpox brought down North in steamers and schooners, during the season of open water. Never that I know of in the winter. Once, after wireless telegraphic communication had been established, I was warned in time, that following the visit of the mail-steamer coming North on the first spring voyage, a

fever was breaking out at each little village where she called. On our advice by telegraph, the captain, who was bringing North some returning soldiers from the World War, routed out 2 sick men on board and made them up beds in the boats hanging in davits over the side, until they arrived at Battle Hospital. We then isolated them, removed them to an empty cottage, and the trouble stopped. But it had to breed much trouble for us, for the sick men violently protested against any such arbitrary arrest on their long journey home, and the people on our island violently objected to their landing on their island, as they were, not unnaturally, in mortal fear. Experience had shown them how very fatal measles and other fevers could be to them, with so little immunity existing.

The law in the early days of our work, insisted on every vessel bringing home in the fall a full complement of her crew, dead or alive. This old English law had some reason in it, for there was no one to certify cause of death, and so avert foul play going unnoticed. The only way this could be done was to salt down the corpses. On one occasion I visited and tried to disinfect a fishing schooner, of which the family were both owners and crew. It was far North, and there was a fair load of fish for market already stowed away in the hold. Under the ballast deck of the after cabin, in which the father and mother lived, were the salted corpses of 3 children who had died of scarlet fever.

Typhoid. There are still no artificial water supplies on the Labrador. Water is drawn from shallow surface wells, unless a house is near a river. In winter, as the ice grows thicker and thicker, water is "hauled" in a draw-buck from an ever deepening hole, and dragged by a train of dogs to the houses. The indescribable mess that accumulates around a well by spring is a serious menace, and we have had several outbreaks of typhoid. We have, of course, put in running-water supplies at all our hospitals, and vaccination has with us, as everywhere, definitely proved its immense values.

Smallpox. Our smallpox has always been of very mild type, and has never, in my memory, been a cause of death.

Diphtheria. Diphtheria has, on the other hand, been once or twice very fatal. On one occasion I was steaming down the Gulf to the hospital and anchored in a harbor called Red Bay, 45 miles from hospital. I visited ashore a house in which a fever had broken out. There being nothing, however, of any severity, I ran across

the Straits to visit a harbor on the south side, while awaiting developments. When I came back a couple of days later, a terrible state of things existed. Terror reigned in the village. No one was allowed to go anywhere near the infected house, and the family of a father, mother and, I think, 7 children, were imprisoned in it at the muzzle of several guns. We had made no diagnosis, but the people told us that there were already 5 dead in the house. Anchoring well off shore, and allowing no more communication with it, we donned operating room dress only, and went to the house. It proved to be diphtheria. We set to work within the cordon, drew out and buried the 5 bodies, and then stripping absolutely naked in turn each of the survivors, we bathed them from head to foot with antiseptic solution, in wet sheets of which we then wrapped them and hustled them barefooted across to another absolutely empty hut, and to new clothing. Yielding to their violent protests I did not burn the house and everything in it, as I should instantly today. But we were then overwhelmed with poverty, and so the small stocks of food, and a few possessions, were spared. The hooked floor mats covered with bloody sputum, however, we piled up outside, and ordered all the stuff thrown out to be instantly burned. A couple of days later two of these mats were found dragged away and hidden "to be washed," as they were considered so precious. Yet the only soap they possessed was made from burned wood ash and seal fat, and they had no antiseptics.

Aboard my boat at that time was a ten-year-old boy going to hospital for eye trouble. Though my shore clothes never went below deck, and my own ablutions were performed under the open heavens, the boy died of smallpox before I got him to the hospital. The epidemic ended where it began. Not one other case developed, and the rest of the family recovered. But the house was never used again, though it stood rotting away for years. In later days, when we were able to keep "one jump ahead of the sheriff," under any such circumstances I always burned the house.

On one occasion this made a very dramatic scene. It was far north in a group of small islands that lies from 10 to 15 miles out in the Atlantic, off Cape Harrigan. We had been called to a tiny hut in which a girl, aged seventeen years, was said to be dying. The coast there is absolutely uncharted. The islands were not even roughly indicated in any way. No one lived in winter upon the island, and only a few summer houses existed besides the schooners.

We had just steamed in and anchored when a Welsh captain of a foreign, three-masted schooner came aboard, and told me that no one but himself had been near the girl for several days, that she was isolated, because no one had any idea what the matter was, except that her abdomen was swollen up hard, and she could not eat anything and had terrible diarrhea. We had to land on the rocks and climb up a very steep cliff. The tiny hut was completely dark, and the girl lay unconscious in a rude wooden cot. She had not been washed or changed for several days, and was on her back half sitting up in a heap of rags and old bedclothes, soaked with the vilest dejecta. The skin of the buttocks was literally rotting off. The captain helped me bravely to remove everything possible. The night shut down with heavy fog, and there was considerable anxiety about a small boat that had not returned with the rest, and in which was the brother of my patient. Stimulants proved useless, the girl never revived, and soon after midnight she passed out. We at once burned the house and everything in it except the body. Oddly enough, it proved afterward that the blaze of the light so high up, had shown over the fog, and enabled her brother to make the harbor as she left it. There is little scientific value, or even unique experience in these gruesome memories, so I will close with but one or two more.

I am asked occasionally about mental trouble. The close relationship of our families may be responsible for some of the brain abnormalities that affect a proportion of our people. It should be easy to do some good work on that, for there are so few complications, and heredity can be so very easily followed. My colleague, Dr. John Little, operated one time on a series of cases of epilepsy. These greatly troubled us under our peculiar social circumstances and we considered these lives a menace, and any effort to avert it desirable, for there is no one to look after or teach such unfortunates. One deliberately burned down, in mid-winter, our large wooden orphanage with 50 children in it. Dr. Little always believed that in one or two of his early cases, which he watched over a period of several years, that they had positively benefited by decompression, although no actual source of brain irritation was ever located. Brain tumors have not been common.

Malignant tumors, except epithelioma about the mouth from irritation by tobacco pipes, bad teeth, etc., are not common, though I have seen, in old days, a man's head eaten off literally by rodent

ulcer. We have possessed for many years the only radium north of Halifax on our Coast, and we naturally get cases from long distances, yet only 11 needed radium treatment this year in our hospitals, out of over 1000 cases.

Neuroses and psychoses, with depressive symptoms, are distinctly rare, considering the terrible anxieties, deprivations and difficulties our people undergo. Divorces, suicides and murders do not occur, and generally our people are placid, contented, quite unlike the conditions of mental equilibrium, among those who, in what we regard as "civilization," and who enjoy rather too much than too little of "things." On the other hand, apparent miracles that might be attributed to supernatural causes are more common by far, I should say, than in what we label "civilization," unless the claims of pagan and Christian shrines can be established. I was bidden once to a "wake" at the home of a fisherman. On arrival I found the feast laid out, the guests "in their blacks" sitting around the room. Everything had been ready for over twenty-four hours, except the corpse.

The patient, a girl, aged twenty-five years, lay propped up in bed, her eyes sealed with pennies, her jaw tied up over the head with a white handkerchief, her arms laid out in death gloves. The last unction had been given hours before. The parents assured me that it would only create needless trouble if I were to examine her, as she was thus already as good as dead. Turning my back to the body, however, I slipped my hand under the night dress and counted a heart beating smoothly at about 72. The priest was consulted and a long pow-wow was held. But any interference was decided against. Next morning, before leaving with my dogs for another village, again I visited the death chamber. Everything was as I had left it, including the feast and the guests; and after listening to a somewhat strangely cheerful goodbye, I left them. Four days later, without having taken food, and without having moved, so they assured me, she suddenly "got up and walked," and she is today a healthy woman.

There is a very distinct superstition regarding all those whose minds are affected, and sometimes they are greatly feared. On one occasion, a large boat drew up alongside my steamer, and proceeded to unload on my deck wooden frames, which are used on the Coast to stretch sealskins out while drying them to whiten in the frost. To our amazement, instead of a seal skin it was a live man,

stretched in the frame. His wrists and ankles were tied out tightly to the corners of the square, and his body with lashings something like those used to hold the skin out. They had kept him there for a fortnight already, and they were really frightened when we let him loose. For lack of space that night, we locked him in the store-cabin over the engine-room, giving him an old feather bed to lie on. When I opened the door in the morning, the weirdest looking patient I ever saw jumped out. He was a dark brown Eskimo with jet black hair, and big white eyes. He had thrown off every rag of clothing and stuffed them down the ventilator, had got himself all over heavy oil and, having split the mattress, had rolled about in the feathers. In the early dawn, as he jumped out, we realized there was some excuse for their treatment of mental troubles.

Of all the epidemics that have taken tax of our people, the influenza that struck this country also so hard, proved far the most fatal. In the absolutely isolated harbor of Okkak, the annual supply-steamer anchored for three days in November. She had 2 men convalescing from flu among her crew. The inquisitive natives swarmed aboard. In three weeks the living had had to give up trying to bury the dead. Out of 365 Eskimo, over 300 were dead, including all adult males. Out of 6 Europeans there, only 1 died. Winter was already upon them, and before Christmas they should have carried the mail on dogs 90 miles south to the next village, of Nain. There was no one to carry it, so the Nain folk came north to find out the reason. After they returned, an epidemic broke out in their village, and a large proportion of their people also perished. The flu found its way south all along our Coast. But no exact records were kept as to its time and method of arrival, and a splendid chance of tracing it was lost.

For years we have tried to induce some hardy research scholar to come and winter in the North among our Eskimos, before they have disappeared, or become too few to make it worth while. He might possibly solve many questions about which one is often asked, such as "Do carnivorous people suffer with eclampsia? What effect has a high-protein diet on malignancy?" While we have always felt that the isolation of possible modes of approach, and the ease with which the advent of a specific trouble can be located should offer valuable sidelights on etiology. The University of New York offered two scholarships of $2500 each for research workers in this field, but hitherto I have failed to procure the students.

Probably the chief scientific information our sub-Arctic country has furnished to the world outside with regard to disease has been through the dietetic deficiencies. A good deal of work has been done on these, among other things. We had been trying to get the people to use sugar instead of molasses as an economic advance, but found that practically molasses was the only article of our diet supplying iron. Professor Helen Mitchell, from Michigan, and Dr. Ayckroyd, from the Lister Institute, were both down studying our conditions last summer, and have reported fairly fully. The Rockefeller Institute also sent down Dr. Smiley two years previously. The new rapid freezing of foods should materially help this problem.

Our first acquaintance with beri-beri was through a Norwegian whaler coming to hospital with all the crew with an edematous multiple neuritis, of which the skipper died. We sent specimens of the viscera to Montreal, and they first suggested it might be beri-beri. After that we noticed a number of cases—the terrible pains and at times the almost complete general paralysis led us to work hard to find out some remedy for it, long before the discovery of vitamins. A family at a small cove first suggested its connections with fresh vegetables. When we made our first visit after the long winter we found father, mother and eldest son practically paralyzed, while 3 smaller children, who always fed on the floor, were perfectly healthy. Potatoes had been the only diet, and they were first peeled or chipped, and the peels thrown to the chickens in the cages around the room under the settles. Inquiry revealed that the children often robbed the chickens and ate the raw peels themselves.

Gentlemen, I must close this wandering essay with apologies for not being able to do better. My plea for accepting your offer is my faith that there is more than one road to truth. We know now that "natural units" limit the capacity of the protoplasm which constitutes our machine for interpreting phenomena as rigidly as it does that of barium chloride, for instance, to the small range of ray lengths that it can reveal. We are becoming more willing to concede the need for, and capacity of, other roads to true science as well as those of chemistry or physics. The views of the metaphysician are listened to with expectation of almost as much practical values as those of the physician, and the good no longer feel obliged to be harsh to the clever, or the clever to be rude to the good. Thus one of my most highly qualified surgical colleagues, on

being obliged to return home, though "without views" that could be filed under the medieval definition of religious, chose to devote his life to enabling poor people in a remote country district in Maine to obtain good surgery without putting it off too long, or being seriously impoverished by being obliged to find it in some large city.

Nor can I close without recording the infinite appreciation by us country practitioners of the patience and generosity of you, the great teachers and leaders of our profession, who permit us to weary you at your clinics, and even encourage us to pirate your discoveries that we may carry new help back to our people. Little you realize, gentlemen, what encouragement this means, when on return we find ourselves able to say to hitherto incurably lame, "Brother, rise up and walk," or to the blind, "Receive your sight." Nor do we ever forget that, so far as these "wounded men" go, the man who stayed at home and kept the inn is at least of equal importance with the Samaritan whose good was attained "by going about."

Mr. President and Gentlemen, I desire to thank you on behalf of myself and the fishermen of the Labrador coast for the generous opportunity you have afforded us tonight to acknowledge the debt our small corner of the world owes to the College of Physicians.

PSYCHOSIS, NEUROSIS AND CHARACTER-DISTURBANCE*

The Nathan Lewis Hatfield Lecture
Lecture X

By FRANZ ALEXANDER, M.D.
BERLIN, GERMANY

The investigation of the dynamic constitution of personality (that is, psychoanalysis) has extended our conception of mental disease to a very considerable extent. The mere description as used in psychiatry could only detect the most pronounced and obvious deviations from the normal, and such description contributes little to an understanding of psychological connections. When we speak generally about understanding another person we mean that we can identify ourselves with him so that we are able to see his subjective difficulties, his feelings, wishes, fears and hopes. We must do the same if we want to understand the pathological personality of a psychotic or neurotic person. Descriptive psychiatry was contented with the statement that in certain kinds of mental disease patients had, for example, megalomanic ideas, while in other disease they were liable to emotional outbursts, and again in others they wasted their time with compulsory ceremonies on getting up in the morning or before going to bed. Descriptive psychiatry did not inquire into the psychological motives of these peculiarities or their connection with the whole mental makeup of a person.

No doubt, to understand an abnormal personality is much more difficult than to understand a normal one. Psychological understanding is based upon identification and identification with another person presumes some similarity. Men understand each other better than they do women, and women understand each other better than they do men. We understand people of Western civilization better than Orientals, etc. The greater the difference

* Read May 21, 1930.

between two minds the greater the difficulty in understanding. We have almost the same difficulty in understanding a savage or a small child as in understanding our psychotic or neurotic patients. The authistic-hallucinatory character of infantile thinking is similar to the schizophrenic mentality, and the mental life of savages has many points of agreement with psychotic and neurotic behavior. They perform, for example, a seemingly senseless ceremonies similar to the peculiar compulsory ceremonies which some of our patients suffering from a compulsive neurosis carry out.

The difficulties of adults in understanding either very young children or savages or psychotics and neurotics have the same reason, namely, that their mental processes are different from the mental processes of normal adults and belong to a more primitive level of mental development.

The most outstanding feature of the primitive mental processes of savages and children is their great capacity to satisfy their wishes in the form of fantasy and illusion, and at the same time to neglect all the external facts which are contradictory to their subjective wishes. They build a fantastic conception of external reality according to their inner needs. To use a technical expression, their mental life is more subject to the pleasure principle than to the reality principle.

Even more interesting is the fact that, just as we do not understand people with this primitive mentality, we do not understand some of our own mental products, for example, the greater part of our dreams: Dreams, as psychoanalysis has shown, are products of a primitive mental activity, and have, therefore, many affinities to such an infantile form of thinking as well as to pathological mental manifestations. A long time ago Bleuler pointed out that dreams and schizophrenic processes possessed a similar authistic character. Since everyone has dreams, the question arises whether we have the right to assume that that part of our personality which produces the dreams is similar to the personality of children, savages and of the insane.

The striking contribution of psychoanalysis has, indeed, been to point out that we really do not understand and do not know one part of our personality, just as we do not understand primitive and pathological human beings. This means that the personality is not homogeneous, since one part of the mental apparatus retains the qualities of the primitive state of development, even after the

change from child to adult. Only one part of our mentality participates in this change, while another part retains the infantile attitude. Whereas in normal persons this infantile part plays a relatively unimportant rôle, psychotic and neurotic persons live more in a primitive state of mental development. To use a technical expression, the greater the fixation of a person is to his infantile situation the greater are the chances for his developing a mental disease.

The important question arises, how does it happen that the infantile part of personality which, according to psychoanalysis, is present in everybody, does not disturb the normal mental processes? If it is true two different persons are living in everybody, namely, an adult adjusted to reality and a child with a mental life subject to the pleasure principle with hallucinatory thinking similar to schizophrenia, how is uniform behavior to be guaranteed? The answer to this question is the second essential contribution of psychoanalysis.

The uniform entity of our conscious ego is guaranteed through a dynamic psychological fact; that is, through repression. One cannot be at the same time an adult and a child. To become an adult it is necessary to forget the infantile way of thinking. The attraction of this infantile form of mental life is very great, since it is subject in a much higher degree to the pleasure principle than the adult mentality, which has to adjust itself to reality. As already mentioned, it is characteristic of infantile mental life that it does not take into consideration the facts of reality which resist the subjective wishes and needs. The recognition of a strange and by no means always benign external reality is the problem which the child has to solve in his later development. The most important means of overcoming the infantile form of thinking and the infantile wishes and tendencies is repression, through which the ego puts away the disturbing remnants of its infantile existence. Through repression these infantile remnants become unconscious and form the unconscious part of personality.

Now we can appreciate better the great difficulty in understanding children, savages and the insane. We cannot understand them, as each identification, on account of the great difference in mentality is difficult, but also because a mental force within ourselves keeps us back from understanding them. I mean repression. To be a normal adult person we have to forget or, in other words, to over-

come the primitive part of our personality, and, therefore, we cannot understand either the primitive mental processes of others or our own dreams, which are manifestations of an infantile personality. Science in the investigation of mental pathology has to overcome the subjective difficulty which is inherent in the fact of our own repressions.

Psychotic and neurotic symptoms can be understood on the basis of the conflict we have just described between the infantile remainders and the adult part of personality. The chief difference between a neurosis and a psychosis is the different extent to which the repressed mental content breaks through into consciousness after overcoming the resistance of the repressive forces. This breaking through of the repressed contents is much more complete in the different forms of psychosis. In the end phases of schizophrenia, for example, one has the impression that the ego has given up all resistance and is dominated entirely by hallucinatory mental processes. In a psychosis even the very first adjustment of the ego breaks down; that is, the capacity of subordinating satisfactions of imagination to the evidence of sense perception. The consequence is a loss of orientation to the world. Of course, all the later results of development, such as esthetic and moral restrictions and inhibitions, also disappear in the psychosis. Psychosis thus can be considered as a flight from reality and from the adult form of existence back to childhood, to a happier time in which fantasy prevails, and does not care for the external facts.

In the different forms of psychoneurosis the battle between the two parts of the personality, between the conscious ego and the primitive id (the technical expression for the unadjusted original part of the mental apparatus) is not decided, since neither of them has a decisive victory and the conflict between them is still manifest. While the end phases of a psychosis can be compared with a silent battlefield after all the soldiers on one side have been killed, in a psychoneurosis the battle is still going on. Psychoneurotic symptoms are partly manifestations of repressed tendencies and are partly reactions of the ego against these tendencies.

From the static point of view, psychoneurotic symptoms can be compared with the outcropping and seemingly isolated points of a big coral-reef which are connected only under the surface of the sea. Neurotic symptoms are similarly isolated indications of a repressed mental content. The conscious ego has still the upper

hand, although it does not succeed entirely in repressing the unconscious tendencies. The important fact which shows the partial control of the ego is that this unconscious mental content can appear in consciousness only in distorted forms. These distortions are the compromises between the two antagonistic forces in the mental apparatus, *i. e.*, they are a compromise between repressed and repressing forces. In these distorted forms the unconscious content can appear in consciousness without hurting the conscious personality. So, for example, peculiar and apparently senseless compulsory ideas which the patient himself does not understand may have an unconscious meaning, expressing some aggressively hostile notions which are incompatible with the dominant feeling of the conscious personality. Psychoneurosis and psychosis can be considered as different stages of the same mental process, that is, of the breaking-through of the unconscious, repressed, primitive part of personality. In a psychosis this process goes much farther, for the difference between the conscious and unconscious parts disappears and the unconscious dominates the whole personality, whereas in a neurosis the principal achievement of the later ego development, namely, the acceptance of reality, remains more or less intact, and the unconscious tendencies penetrate the ego only in isolated symptoms which play the rôle of foreign bodies imbedded in normal tissue.

I am aware that this representation can not give you a plastic picture, being an abstract and concentrated extract of the facts. I would give you some examples as illustrations, but the emphasis of my paper is on the third form of mental disturbances, that is, on pathological characters. The investigation of disturbances in the character is the latest accomplishment of psychoanalysis and it is not yet so much discussed as the psychology of neuroses and psychoses. These pathological characters have no pronounced symptoms, and yet even lay people who have to do with them feel that they are not normal. I venture to say that descriptive psychiatry, as long as psychoanalysis did not supply it with a kind of mental microscope, did not know much more about these persons than the laity. These psychopathic personalities—this is the technical expression in Germany for them—were a neglected chapter of psychiatry containing only some general statements. Different authors divided them into various groups. Thus Krappelin speaks of extravagant persons—tramps, dipsomaniacs and gamblers, Bleuler, distinguishes different groups, such as irritable characters,

unstable personalities, queer fellows and enemies of society. To these groups belong, also, those who were formerly called morally insane. Significant for these types is that they cannot be put into the well-known nosological groups of psychoses and neuroses. Apart from the fact that one cannot make a clear diagnosis of them, another outstanding feature is their high grade of intelligence. Only the emotional life and behavior is disturbed, the function of judgment and the capacity for logical thinking remain intact. This is the reason for expressions like "emotional feeblemindedness" or "moral imbecility." Also, they behave irrationally, as if their intelligence was not normal; intelligence tests, however, often show a hypernormal intelligence and, furthermore, pronounced capacity for moral feelings and judgments. The psychiatric diagnosis of these psychological types was very uncertain and was based more on intuition than on a knowledge of the mental processes characteristic of them.

This widespread group of paradoxical characters includes the most different types of personality. To these belong, besides some delinquents, all kinds of adventurers and gamblers; but, also, more harmless types, as queer, original fellows, possessed collectors, foolhardy sportsmen, daring tourists, etc. Thus we can distinguish a socially harmful from a socially harmless group, the delinquents from persons with irrational but not antisocial passions. This second socially harmless group I should propose to call eccentrics, which corresponds to the French expression, "Originale," and to the German, "Sonderling."

A deeper understanding of these paradoxical characters was available only after the emotional basis of human behavior was investigated by the methods of psychoanalysis. In these characters the unconscious repressed tendencies do not manifest themselves in isolated symptoms as in neuroses, but they influence the whole behavior, the whole current of life.

In contrast to neurotics and psychotics, these personalities are extraverted, vigorous types who are not contented with an unreal that is fantastic satisfaction of their unadjusted infantile impulses. They do not produce symptoms, but, instead, live out in action identically the same tendencies which neurotics and psychotics express in their symptoms. Consequently, their whole life has a dramatic character and is influenced by the repressed primitive part of personality. The impression they make at first sight is

just the opposite of that which neurotics make with their symptoms. The greater part of the mental energy of the latter is absorbed by their symptoms, and, therefore, neurotics are mostly inhibited and inactive personalities. They have a rich inner life of fantasy and on account of this their real life and actions are restricted. One could express this difference by saying that neurotics are the negative of these abnormal characters, which we call *neurotic characters.*

There are three characteristic signs which one finds to be typical of this kind of pathological personality: the first is a certain *irrationality* in their behavior; the second, a *stereotyped repetition* of these irrational acts and, third, the presence of *mental conflict.* All three signs are the consequence of the same psychodynamic situation, namely, of the preponderant influence upon behavior of unconscious motives over those of the conscious ego and its rational logical judgments. The unconscious motives which are not adjusted to reality are responsible for the irrational trend in behavior. Also, the stereotyped repetition of eccentric acts have the same basic cause. These people do not learn from experiences and do not consider external conditions because their conscious ego has not a corrective influence upon their actions. The instinctive motives come to expression in spite of better judgment, and this gives to the actions a compulsive and stereotyped character. These two features, irrationality and stereotyped repetition, impart to the life of these people a demonic and fatalistic quality described by Freud in his work *Beyond the Pleasure Principle.*

The third sign is the *mental conflict* which casts over these lives a tragic shade. Conflict arises because, as we have already pointed out, the intelligence is normal and the ego is intact, but powerless in the face the urge of repressed forces. After the deed regret comes too late. These people act against their own conscious wishes and feelings, for they are compelled to carry out irrational actions as if they were possessed by an evil demon. A large number of delinquents belong to this group, which, therefore, possesses a great importance in forensic medicine. These people make up their minds again and again to reform and begin a new life, but they never can carry through this conscious intention, for the influence of their conscious personality is less than that of their unconscious.

Especially interesting are the cases in which it appears that no conflict and no conscious regret or condemnation of this compulsory

behavior is present. A deeper psychoanalytic investigation always shows that even in these cases a conflict is there in the unconscious. A deep, unconscious wish for punishment, which is a consequence of the conflict, can be detected, and this manifests itself in self-destructive tendencies. These self-destructive tendencies can betray themselves in most different ways, the most extreme form of which is suicide. In other cases their tendency to punish one's self results in destroying own successes, or leads to putting one's self in a difficult situation, or, in the case of criminals, in committing unconsciously purposive errors which are liable to betray the perpetrator to the police.

The psychological connection between the compulsive asocial actions and the self-destructive tendencies which exhibit a moral origin was not easy to find. A primitive theory of criminal justice appears in these mechanisms, the gist of which is that punishment or suffering is able to expiate crime. This mentality, which still prevails in primitive civilizations, is the principle of Talio, which considers punishment and crime as two different currencies which can be exchanged at a certain rate. I was able to observe this type of mentality in a young Catholic patient of mine, who was reared in a strict convent. The adolescent girls had to confess their sins in weekly confession. My little patient had a peculiar method of gaining for herself an advantage from these weekly confessions. She confessed sins—for example, having read some forbidden books—which she had not yet committed and so received absolution which she applied to the coming week, when she read the books with an untroubled conscience. In this way she paid in advance the price for the forbidden act. The same psychology is the basis of the paradoxical coëxistence of asocial or even criminal tendencies and self-punishing reactions in neurotic characters. Although they have a well-developed conscience which condemns their antisocial behavior, through self-punishing acts they are able to bribe their conscience and commit actions which they otherwise would condemn. They pay for it through self-punishment or real punishment which they themselves provoke. Punishments have with this kind of characters a paradoxical reaction. They do not inhibit; on the contrary, they favor the antisocial behavior, disarming the moral inhibitions and relieving the sense of guilt. The tendency to expiate one's own conscience, or to expiate the projected form of the conscience that is God, through suffering, is deeply rooted in human

nature and is the basis of ascetic tendencies in religion as well as of sacrifice.

Dangerous situations have for the more harmless types—such as adventurers, gamblers, daring sportsmen—the same significance as unconsciously provoked punishments have for delinquents. These characters betray the same sense of guilt as the neurotic delinquents, and the uncanny tendency to risk their lives to play constantly with death has the deeper purpose to expiate their conscience and to get rid of the unbearable sense of guilt. The origin of this sense of guilt in these cases is not so transparent, because their actions, their hobbies and passions, though irrational, are not antisocial, as are the actions of delinquents. A deeper investigation, however, proves that all these hobbies cover up similarly asocial tendencies, although milder forms than those of the delinquents. These people are not antisocial, yet asocial; they have a hobby which has no social value, which is a kind of private occupation; they are not enemies of society, but they despise society; they create a life of their own which has no connection with the aims of community.

Psychoanalytic investigation proves that both the antisocial attitude of delinquents, as well as the peculiar irrational predilections of the eccentrics, are rooted in the same infantile conflict, in the son-father situation. Both types, the antisocial delinquents and the asocial eccentrics, could not solve this important emotional situation of their early childhood.

It is not possible for me in this lecture to represent the validity and importance of this conflict, and I must content myself with referring to the fact that the attitude of rivalry toward the father is one of the great emotional problems of every little boy. This feeling producing an unsolvable conflict mostly becomes entirely repressed. The conflict arises because in the same time the father is the object of positive feelings as love, admiration, etc. These ambivalent feelings are responsible for the fact that most boys repress the negative hostile part of their relation to their fathers. Having them so deeply repressed, most people never can remember these hostile feelings of childhood except through a thorough analysis.

Apart from the repression, another frequent solution of the father conflict is the transference of the hostility to other objects which involve less conflicts.

Thus delinquents, for example, through their whole life are fighting

the social order as a successor to the father, while the nonsocial eccentrics avoid the free battle, but in their hobbies we can detect an interestingly distorted form of their hostility against the father. In their unsurpassed records in their uncomparable collections in the unscaled mountain peaks they conquer the father, that is, they try to solve the emotional conflict which a small boy feels toward his powerful father.

It is easy to understand why the delinquent belongs mostly to the lower classes, while the eccentric is mostly the son of a good family. In the case of neurotic characters of a socially low position it is very easy to transfer the hostile feelings felt toward the father to the state and society in general. The society which has treated him badly gives him a good, rational motive for these transferences of affect; whereas, the eccentric enjoyed a happy youth, good education and, later, the whole world is open to him. He has no real difficulties, he has no obstacles in society. Therefore, he has to create new objects for his hostile aggressions; a life of his own with its own problems, ideals, aims and difficulties, the overcoming of which enables him to relieve the son-father conflict of his childhood.

Thus we can trace the difficulties of later adjustment back to the emotional problems of youth. Both the antisocial delinquents and the nonsocial eccentrics, the two big groups of maladjusted personalities, are victims of these similar conflicts of their childhood.

Another typical family situation was first pointed out by Abraham and is common in the life histories of adventurers and swindlers. In families with many children the youngest boys often exhibit a certain instability and inclination for adventure. Such a boy has not only one competitor in the father, but also a number of older rivals in his brothers, and consequently, has the most difficulty in excelling and in drawing attention to himself. He is submerged in a big family and is sentenced to a drab and undistinguished existence. Furthermore, he is very often an unwanted child, and his birth due to lack of foresight or precaution. He has a long time to wait to share of importance and attempts to achieve it by reckless sensational behavior. The first-born son has a much easier time, for he is most favorably situated; can afford to be correct, patient and respectable; to do the right thing and become an orderly citizen. The youngest, if he wants to surpass his older brothers, has to accomplish something extraordinary. This impatience and dissatisfied exhibitionism of childhood is often the basis of the careers of typical

adventurers. Their scandals and extravagant affairs are the conspicuous acts which they perform in order to satisfy their overstrained ambitions and to become an object of general interest.

These are only some samples to demonstrate how the later development of character is determined by the early family situation—that is, through the emotional conflicts of family life. The old conception of psychiatry, which considers character as something static and determined only by heredity, is untenable in face of psychoanalytic experience. Many pathological cases, diagnosed formerly as psychopathic constitutions, acquired their character during their postnatal development.

It is natural that not only the formation and the destiny of pathological characters, but also the development of normal people, involve the same determining factors. Psychoanalytic investigation proves more and more that what we call destiny is not only the end-result of inborn character and later accidents, but also the consequence of the emotional problems of early childhood.

The important practical consequence of these facts is that the problems of influencing destiny and character is no longer a divine but a human affair—namely, a problem in therapy. The pathogenic influences of postnatal existence can be corrected through the emotional experiences of a psychoanalytic treatment. These influences, being indeed of a psychic nature, can be neutralized by other psychic experiences—that is, by psychotherapy. Character analysis is consequently a psychotherapeutic problem *par excellence.*

The prospects for the psychoanalysis of pathological characters those abnormality is not chiefly constitutional are even better than the prognosis for neurosis treatments. It has already been mentioned that those abnormal characters which exhibit no pronounced symptoms stand in one important respect nearer to normal persons than the neurotics, namely, that they are not contented with the unreal and fantastic satisfactions which neurotic and psychotic symptoms can afford, but need real satisfactions produced by real actions. Their impulses and instincts do not incline to introversion, like those of neurotics, and, moreover, they have a markedly expansive character, as that their life is often very dramatic, eventful and adventurous, in contrast to those of neurotics and psychotics, in whose lives fantasy plays the predominant rôle. The chief dynamic difference between psychotics and neurotics, neurotic characters and normal persons, can be expressed by the

following scheme: Neurotics and psychotics stick to the infantile forms of their mental life and do not adapt themselves to reality, so that they are forced to take refuge in the realm of fantasy, where they can imagine whatever they like. They sacrifice real satisfactions and content themselves with substitutes provided by their fantasy and symptoms. Normal personalities change the infantile quality of their mental processes and adjust them to the demands of the external world. Thus they sacrifice the original satisfactions of childhood, but gain in exchange the real satisfactions of adult life. Psychopathic characters would like to have both what neurotic and normal people have; they wish to keep the infantile forms of satisfactions and at the same time are not contented with fantasy, but wish real satisfaction. Consequently what distinguishes neurotics and psychotics from normal persons and from neurotic characters is a biologically basic feature of the instincts; that is, the tendency to intravert satisfactions into fantasy and to renounce real satisfactions.

But it is especially difficult to influence by means of psychotherapy the inborn quality of instincts, and notably the inclination to introversion—that is, the tendency to give up action for fantasy. This difficulty, however, does not meet in the case of character disturbances, as they have the same expansive and extraverted kind of impulses and instincts as normal persons, and only the coöperation between instincts and conscious ego is disturbed. To overcome these disturbances is, no doubt, a problem which lies within the possibilities of psychotherapy, and I, therefore, venture to say that they are the most appropriate objects of psychoanalytic treatment. The principal difficulty, however, is that such people are not so likely to submit spontaneously to treatment as persons with pronounced symptoms. This is especially true when they are young and have not as yet suffered much from their compulsive character traits. Being extraverted types, they are cheerful and vigorous personalities, and do not realize their own abnormalities. The internment of such cases, therefore, in institutions for psychoanalytic treatment is highly desirable. With these characters it is the same as so often in medicine: Those whom you can help do not come to you, and those who do come, come too late.

REGENERATION REACTION TO SULFHYDRYL AND ITS PARTIALLY OXIDIZED DERIVATIVES*

By FREDERICK S. HAMMETT, Ph.D.

(The Research Institute of the Lankenau Hospital)

The work at the Research Institute of the Lankenau Hospital has uncovered the fundamental biological fact that growth by increase in cell number is regulated by a chemical equilibrium in which the sulfhydryl group and its partially oxidized derivatives are the constituent elements. Cell proliferation has been stimulated in plants, paramecium, rats, mice and man by a variety of compounds bearing the $-SH$ group, while retardation has been effected in these several forms by the $> S = O$ or sulfoxide group.

In view of the biological kinship between malignancy and the phenomenon of regeneration as exhibited in simple organisms, a study was made during the past summer of the reaction of the latter to the significant chemical groups.

The regenerating first right foot of the hermit crab (*Pagurus longicarpus*) was used as the test object of the results recorded here.

The details of the procedure as to the care of the animals and experimentation as worked out by my coworker, Miss Dorothy Wall Smith, will be reported in another place. All that is necessary to tell here is that the crabs were kept in fingerbowls in fresh seawater to which the chemicals were added in the desired concentrations, and that the durations of the trials were seven and fourteen days from the time of removal of the foot.

It was consistently found that 5×10^{-7} sulfur as R–SH in p-thiocresol stimulated regenerative growth and differentiation, while 6.5×10^{-7} sulfur as $\frac{O}{R\text{–}S\text{–}R}$ in di-phenyl-sulfoxide resulted in marked retardation. Appropriate seawater and chemical controls were run in parallel with the test cultures.

These results, therefore, extend the principle of the chemical

* Read October 1, 1930.

regulation of cell proliferation to another animal form, to another milieu (seawater) and to another type of expression of growth by increase in cell number.

They, further, give presumptive evidence on a biological basis for the assumption that malignant growth, like all other so far tested, is significantly conditioned by the sulfhydryl-suboxidized sulfur equilibrium.

This work was done at the Marine Experimental Station of the Research Institute of the Lankenau Hospital. It was made possible by Dr. Stanley P. Reimann, Director of the Institute, and through the generosity of Mr. August Bein, of Philadelphia, and the Captain L. D. Baker Estate, of Boston.

HEPATITIS*

By JOHN B. DEAVER, M.D.

The surgeon is often called upon to wander in the upper abdomen and find the cause of the patient's failure to get well under medical treatment. Not infrequently, in this study of the pathology of the living, he finds what has been described as hepatitis. While the term "hepatitis" is, perhaps, not well chosen for certain conditions, for others it describes the state of affairs with considerable exactness. Just what is included under the term hepatitis will be described later. In order to make the finer distinctions in this form of pathology of the liver, it is the custom in our clinic to consult with the staff, particularly with the pathologist, and thus obtain an interpretation of the lesion and its background from the pathologist's point of view. Besides this, the pathologist is often given a sliver of liver removed in taking out the gall-bladder and thus the gross interpretations can be checked up by microscopic study. It goes without saying that not only is a correct interpretation of the pathology obligatory, but it is essential to try to trace the possible origin of the pathology. With the abdomen open, this entails a careful survey not only of the liver, the gall-bladder, the extrahepatic ducts and their lymph nodes and the nodes in the gastrohepatic omentum, much in evidence in some cases, but also the duodenum, the stomach, the spleen, the pancreas, the appendix, the large and small intestine, especially the sigmoid, and the pelvic viscera; in short, a complete autopsy *in vivo*.

The increased attention accorded the subject of hepatitis in recent years is principally due to the desire to prevent biliary cirrhosis, the terminal stage of a chronically infected liver. I have been much interested in this condition for some time and have stressed its importance in various articles and addresses.

As in all subjects of discussion, increased and careful observations uncover more and more instructive examples, and thus, as

* Read October 1, 1930.

experience piles up, add more details and greater clarity to the picture.

The first point to consider is the etiology of this form of hepatitis. It is frequently associated with gall-bladder disease, not only qualitatively, but very often quantitatively. The quantitative aspect, however, has two phases, the duration of the infection and its severity. It is not uncommon in chronic disease of the gall-bladder of long standing to find fairly extensive changes in the liver. On the other hand, in cases of shorter duration, the changes may be confined to an area in the immediate neighborhood of the gall-bladder. How are these changes brought about?

I have frequently spoken of gall-bladder infection, its manner of occurrence, the difficulty of eradicating it, its persistence and its chronicity. I have, also, repeatedly called attention to the relation of the appendix to gall-bladder infection, and have emphasized the rôle of the appendix in the pathogenesis of hepatic infections. This subject of hepatic infection is not a new one. As far back as the 90's the great pathologist, Adami, in sections of liver found organisms within its substance, to which he gave the name "subinfection." We now call it "focal infection." We know what an ideal location the appendix is for lingering infection because of its narrow lumen, the difficulty with which it empties and its rich lymphatic supply. Its venous system, being that of the portal vein, adds to the relationship in providing a possible path for organisms to reach the liver. I am not the only one who feels that an abdominal operation, especially one for chronic infection, is incomplete without the removal of this pernicious source of infection, the appendix.

Now that surgeons in their autopsies *in vivo* have demonstrated the close association of cholecystic disease and changes in the liver, the experimental work of research laboratories has added materially to our understanding of how this infection is carried. In the first place, it has been shown, over and over again, that streptococci, once in the walls of the gall-bladder, are likely to persist there for an indefinite period. In the second place, even with infection in the walls of the gall-bladder, the lumen of the organ is not necessarily infected. Our knowledge of the anatomy of the gall-bladder shows us that it is an extremely vascular organ and one with an abundant lymph supply. As I have previously stated, in dealing with the subject of the gall-bladder itself, the lymphatics of the wall of the

organ communicate with those within the liver itself. Years ago, pathological reports on bits of liver tissue excised during cholecystectomy already showed that when the gall-bladder was diseased the liver was never free from changes, varying from cloudy swelling and fatty degeneration, to fibrosis of the portal spaces, to actual chronic inflammatory proliferation and exudation. Now the work of Graham has demonstrated this direct connection experimentally, while Wilkie's researches emphasize the connection, adding the possibility of still another route of infection. It is quite probable that interstitial infection of the gall-bladder and the consequent fibrosis and hardening cause at least some obstruction, if not complete blockage of the lymphatic communications between this organ and the liver itself. After separating the gall-bladder from its bed, Wilkie found that the intravenous injection of streptococci was often followed by cholecystitis, and that when the gall-bladder was allowed to remain in its normal situation, similar intravenous injections produced infection of the liver. The trauma of this experiment probably precludes the drawing of conclusions as to normal physiology, but it decidedly imitates the pathological separation of the gall-bladder from the liver as it occurs in long-continued fibrosing inflammations.

Picture, then, this chain of events: the gall-bladder is infected, perhaps by way of the appendix, and the walls of the gall-bladder become a convenient nesting place for organisms active and inactive. They enter the liver. When active, they spread rapidly; when quiescent, small amounts of connective tissue are constantly being laid down to wall them off. The part of the liver adjacent to the gall-bladder now being infected by very reason of its anatomy allows the spread of the organisms throughout its entire structure, for the capsule of Glisson sends prolongations and divides the organ into lobules, so that there is a direct communication by way of the lymphatics from one end of the organ to the other. The infected liver then excretes infected bile, which in turn leads to cholangitis and cholecystitis. At the same time, organisms occasionally may enter the blood stream, and intravenous injections may reach the gall-bladder to damage it still further and thus again around the vicious circle. Naturally the whole body shares in the toxemia. As concrete examples of this, culture studies in our own clinic give the following results:

Bacteriological Findings of Two Hundred Gall-bladders

Pathological findings of 200 gall-bladders.	Number.	Positive.	Positive per cent.	Streptococcus anhemolytic, per cent.	B. coli, per cent.	Staphylococcus albus, per cent.
1. Acute suppurative and ulcerative . . .	10	6	60	50	17	33
2. Chronic interstitial and catarrhal	86	42	48	43	28	28
3. Chronic suppurative and ulcerative . .	7	3	42	67	33	
4. Minor lesions, chronic atrophic	97	29	29	31	7	62
Total	200	80	40	42	16	35
Hilus lymph nodes .	200	72	36	41	18	40
Calculi in gall-bladders	66	38	57	39	26	28
Bile in gall-bladders .	200	39	19	41	36	22

The gall-bladder, its contents and the lymph node at the root of the cystic duct have been studied in relation to the organisms present. Naturally, it is recognized that the mere presence of organisms does not mean infection; the lymph nodes or the tissues may be perfectly capable of destroying them without undue harm to themselves. Nevertheless, under ideal conditions it is probable that no organisms at all will be present; but no one can predict what they may do in the future if they are present and persist or are continually being brought to the liver.

To return to our subject, a very common finding is a thick, heavy liver with the anterior edge rounded, the capsule tense, the parenchyma swollen and somewhat fragile, The color varies from a yellowish-brown to a dark reddish-brown or a bluish-brown. Strictly speaking, this is not hepatitis, but a cloudy swelling and congestion. When the gall-bladder is thickened, cloudy and opaque, and has lost its normal tonus and elasticity, more especially when the lymph nodes in the neighborhood are enlarged, the gall-bladder should be removed, for it is infected and some change in the liver is to be expected. Cloudy swelling and congestion are recoverable lesions. This is especially true of the liver, which has a large power of recovery, and the removal of the gall-bladder, the most important link in the vicious circle of infection (naturally the appendix also), brings happy results. With reasonable after-care as to exercise

and diet, a complete recovery is assured in practically all cases. The more acute the infection, the more advanced will be this particular change, until the actual spread of organisms causes adhesions to the omentum and the outer and posterior abdominal walls. In long-continued cases of milder degree, adhesions are fibrous from the beginning and may finally involve also the hepatic flexure, the duodenum and the stomach. This large, thick, heavy liver, therefore, is found more often when the gall-bladder alone is infected than when the common duct is involved, in which condition the lesion in the liver is apt to be more advanced.

Real hepatitis in the pathological sense—that is, inflammation of the liver with exudation—occurs in a localized and a generalized form.

As stated, acute inflammation in the gall-bladder or anywhere else produces a cloudy swelling and other degenerative changes. But in the special case of the gall-bladder there are added the insults of close anatomic proximity through lymphatic and physiological contact through the bile. Thus we see more or less exudation of serum, fibrin, polymorphonuclear leukocytes in and around the gall-bladder bed radiating into the liver. If of sufficient duration, the infection travels from this region over a large area of the liver. Gaining access to the bile, both upward from the gall-bladder and downward from the liver itself, the infection then travels up and down the ducts to all parts of the liver and finally produces a suppurative cholangitis with multiple abscesses and practically certain death. But these cases, important though they are, seldom cause argument, for their treatment is definitely surgical, their course is definitely acute and the symptoms definitely diagnostic. Their prevention, however, is another matter. Broadly speaking, it is the subject of this paper.

It is with two other classes of cholecystitis that we must deal more fully because, after all, such acute, fulminating, gall-bladder suppuration is uncommon compared to the chronic infection and the chronic infection with subacute exacerbation. In chronic cholecystitis the changes in the liver, when localized, show superficially infiltrated opaque areas around the gall-bladder bed, with opaque lines radiating in various directions, together with slight enlargement of the liver as a whole; or there may be a small, superficially infiltrated, opaque patch on the anterior surface of the right lobe of the liver immediately overlying the gall-bladder, the liver

being otherwise normal. This progresses to opacity involving practically all of the anterior surface of the right and, to some extent also, the left lobe. The infiltration into the substance of the liver is manifested by its firmness to touch as elicited by bimanual palpation. The spleen may or may not be enlarged. Finally, there is a small, contracted, firm liver, its anterior border being wedge-shaped, its edges thin, opaque and contracted. The spleen in such cases is firm and enlarged and on microscopic examination is fibrotic.

A variation of probably another stage is a very large liver extending down to or near the crest of the ilium, with a very irregular, nodular surface, the elevations to superficial examination being not unlike cysts and yet of the same consistency as the liver. Microscopically these nodular areas are usually areas of regeneration. I have never seen this type of liver without involvement either of the common or the hepatic duct.

Since it is the lymphatic and the biliary systems which are involved in this type of liver, fluid in the abdomen does not occur. Nor is jaundice common except in the case of the nodular liver just described.

As the lesion progresses, then, we find with increasing frequency common- and hepatic-duct pathology, duodenal or gastric ulcer, chronic or subacute pancreatitis, usually of the head of the organ. Naturally, the appendix and the gall-bladder are both diseased. I cannot help regarding the appendix as the most common site of intraäbdominal infection from which these troubles may arise. The teeth, tonsils, etc., play a small rôle in this state of affairs. On the other hand, the colon may be of importance—not so important as the appendix—but, nevertheless, a factor at times in the production of hepatitis. The question, are these types of hepatitis due entirely to biliary-tract infection? is most pertinent. The answer is that biliary-tract infection does produce these types of hepatitis. It is possible that other causes, probably metabolic in nature, are occasionally forerunners of a biliary cirrhosis, but, I venture to say that, even when begun by metabolic faults, the altered physiology would certainly be conducive to and favor infection.

Is the infection more often lymph-borne than blood-borne? This question is difficult to decide, but from the foregoing, it would seem that at least in the beginning, the lymph channels are more important routes than is the blood stream.

All of these forms of hepatitis are the forerunners of biliary cirrhosis. That seems clear. By biliary cirrhosis we mean, natur-

ally, not the hypertrophic biliary cirrhosis of Hanot, but the fibrosing process—beginning in and spreading from the ducts.

In the last stages of biliary cirrhosis—which are seldom seen by the surgeon, not only because the condition is too late for surgical help, but because it is regarded as strictly medical—ascites is uncommon. Such patients travel straight down hill from chronic toxemia, both from the infection and the loss of liver function.

To summarize: Acute attacks and chronic, and chronic with recurrent acute or subacute attacks of infectious activity in the gall-bladder region are forerunners of hepatitis, varying from purely degenerative changes to a mildly acute, suppurative inflammation to chronic fibrosing, specific tissue-destroying processes, leading to early death from sepsis or to late death from toxemia, and loss of liver function preceded by prolonged invalidism.

As far as treatment of hepatitis is concerned, it is both operative and non-operative. It is quite obvious that operative treatment may be preventive or curative. To be preventive it must be instituted early, before the infection has traveled on its devious roads. This means that removal of the gall-bladder alone ordinarily suffices. But when the infection has reached the liver, cholecystectomy is not sufficient. Nothing short of removal of the gall-bladder, together with *T*-tube drainage of the common duct, will answer. If the latter is carried out comparatively early, it may still be preventive and head off a peripancreatic lymphadenitis, as well as other sequelæ. If common-duct drainage is not instituted until late, it may have to be continued for months.

But what about the questions left unanswered or answered with hesitation? There is nothing to say but that a question unanswered should be a stimulus to a hunt for its solution, usually to be found in clinical and laboratory study, for very few can be answered in the clinic alone, and not so many in the laboratory alone. Unfortunately, the work done in the laboratories of most of our hospitals is simply routine and hunters are few. It goes without saying that routine work must be done, and must be done quickly and accurately, but what is needed in most laboratories is the little bit more which attempts to answer unsolved problems. There is no escape from the conclusion that an operating-room suite is incomplete without a laboratory equipped to do research work. We must educate our philanthropists to the point that they never endow an operating room alone, but complete the gift by adding an experimental laboratory with funds for its upkeep.

DISCUSSION

Dr. James M. Anders: I wish to express my appreciation of Dr. Deaver's splendid presentation. Doubtless, all clinicians are in agreement as to the mode of origin of the majority of cases of hepatitis that Dr. Deaver has so well described. That is to say, they are secondary to infections of the appendix, often of the appendix and gall-bladder together and other adjacent organs. Medical clinicians recognize another definite group of cases of hepatitis, which is of bacterial origin and due to the introduction of microörganisms through the general circulation, and when these cases come to autopsy, as they may, considerable damage to the liver tissue may be observed. A very interesting fact is that these specific organisms have a selective affinity for certain little groups of cells in the lobules of the liver, and this fact is very difficult of explanation. I was very glad to hear Dr. Deaver's remark that the liver had great recuperative power in itself. I think that fact should be well emphasized and is becoming more widely appreciated and better understood. Considerable mechanical injury to the liver is often speedily recovered from. The injury done by chloroform is also frequently recovered from with remarkable rapidity. On the other hand, Opie, in a series of experiments, introduced with the chloroform, bacteria, and found the damage was greater than from chloroform alone, and also that in these animals the healing progressed more slowly. I think the College is to be congraulated upon a paper of such great interest and decided significance as that presented by Dr. Deaver.

BACTERIOLOGY, ETIOLOGY AND SEROTHERAPY OF INFECTIONS OF WOUNDS (TRAUMATOSES), ESPECIALLY OF GAS GANGRENE*

The Mary Scott Newbold Lecture, Lecture XXIV

By PROF. M. WEINBERG
PARIS, FRANCE

I beg you to accept my sincere thanks for having invited me to speak about the researches of my laboratory on the infections of wounds and on their serotherapy. It is for me at the same time a great honor and a great pleasure. It is of course an agreeable task, the work being concluded, to retrace the steps of an immense effort exercised not only by my laboratory, but by the bacteriologists of every front during the years 1914–1918. The study has gradually lifted the veils which completely covered some aspects of the surgery of war, which are equally interesting in time of peace on account of the numerous accidents occurring in ordinary life.

When war broke out, very little was known about the clinical symptoms of gas gangrene and the microbic agents which regulate its evolution. Classical literature described only one form of that infection, locally characterized by a gangrenous wound, the color of which was between red and dull-gray, the odor of which was unbearable and nauseous; the wounded limb appeared tumefied, the skin became covered with bronzed patches and with phlyctenes of bloody content; the final characteristic was the crackling sound heard at palpation round the wound and at the tumefied tissues.

From the bacteriological standpoint, only two anaërobic microbes were known to act as causal agents of gas gangrene, V. septique and B. welchii.

From the beginning of the war, during the months of August and September, 1914, a very great number of wounded soldiers came to Paris with symptoms of gas gangrene. A certain number of the wounds were of the classical type, but others were disconcerting to

* Read, November 5, 1930.

the surgeons through their symptomatology. Nevertheless, these latter, which proved to be extremely grave, were also mentioned under the name of gas gangrene. Confusion did not disappear before a comparative study had been made of the clinical syndromes and the microbic flora in each case.

* * * *

In September 1914, Colonel Cummin, of the British headquarters, impressed by the great number of deaths caused by gas gangrene among the wounded, found it necessary to take prophylactic measures. He applied to Professor Roux, director of the Pasteur Institute, asking him to send one of his collaborators to make a bacteriological inquiry on the spot. Dr. Roux entrusted me with this mission. I went to the front and it was there that I observed the first cases of gas gangrene. My first bacteriological researches were made on the material gathered in the British military hospital in Versailles. Later, charged by the French government to study this infection, I studied many cases in different military hospitals, both in the zone of the interior and at the front.

Very rapidly, I was followed by a very great number of bacteriologists from every country engaged in the war. I have already had the occasion to make, as impartially as I could, an analysis of all the publications on the subject, first in "Traité de la gangrène gazeuse," then in "Données récentes sur les microbes anaérobies." As the time at my disposal is short, I shall take leave to mention only the more important researches, indispensable to the explanation of my ideas.

I propose in my lecture to follow the subject under different headings: first to remind you of the pathogenic microbes which have been isolated from wounds; next to mention the influence of their associations on the evolution of the infection; then to describe the comparative study of the microbic flora with the different clinical syndromes, which will allow us to establish a classification of the different infections and finally, the experimental serotherapy and its application to man.

From the beginning of our bacteriological researches we were struck by the following fact. At first we could not find in the wounds of serious cases any pathogenic anaërobic microbe other than B. welchii. V. septique was not recognized, in spite of very many studies, before March, 1915, eight months after the outbreak of the war. This fact cannot be explained by defective experimental

technique; no other bacteriologist observed this anaërobic species earlier in the war than we did. Moreover, later on, when the complete study of microbic flora had been achieved, we again took up the study of the serous fluid of the cases we have been mentioning which had been kept in ampoules on ice, and the absence of other anaërobes was confirmed.

We are bound to explain this fact by the evolution of the war itself; as the struggle went on, the soil in which the trenches had been established became far richer in microbic species, on account of the constant accumulation of human feces and of the dung of animals accompanying the troops.

I shall not give any details concerning the pathogenic microbes isolated in the cases of gas gangrene or in gas phlegmon; I shall only mention such characteristics as are necessary to understand the evolution of the wounds in which they are to be found.

B. welchii is almost always pathogenic and produces a lesion in the guinea-pig, very often with the same evolution as in man. In this film you see the characteristic lesions, reddish wound, subjacent gangrene, tumefied limb, bronzed patches and hemorrhagic phlyctenes.

Here is another film of a gas phlegmon caused in the guinea-pig by an intramuscular injection of a mortal dose of B. welchii. You see that the muscles of the thigh are completely destroyed at the level of the injection; this phenomenon is explained by the proteolytic properties of B. welchii, which are far more pronounced in a living organism than in artificial cultivations *in vitro*. Besides, gases which have escaped when the phlegmon has been cut through as well as the hemorrhagic edema are due to the special gas-forming and strongly hemolytic properties of this microbe.

It is interesting to note that this anaërobe does not always show its gas-forming character; it sometimes happens, though rarely, that it does not secrete anything but a nonhemolytic toxin. In this event, an injection under the skin or in the muscle of a guinea-pig, causes formation of edema without evolution of gas.

Before the war it was thought that V. septique was the pathogenic agent the more frequently found in gas gangrene. We can say that during the war it occupied only the third rank and was seen in only 10 per cent of the cases. We mention it just after the B. welchii to have done with the known species.

V. septique causes in the guinea-pig, as in man, a gelatinous

and reddish edema in the subcutaneous tissue and in the muscles, with gas-emission. However, it sometimes happens that the anaërobe produces a toxic edema without any trace of gas infiltration; we have seen such cases in guinea-pigs and in horses. The muscles are not destroyed; that is to say, V. septique has not the slightest proteolytic property. But it is strongly hemolytic; it produces also a strong toxin, sometimes able to kill a rabbit of 2 kg. by an intravenous injection of $\frac{1}{10}$ and even $\frac{1}{20}$ cc. In the guinea-pig, far more often than in man, small phlyctenes of the skin, with hemorrhagic content, can be seen.

In order of frequency, B. œdematiens comes next to B. welchii among the anaërobes involved; we found it in a third of the cases. It is a very toxic and very pathogenic microbe. It resembles very much B. novyi, but it is not identical with it; I have therefore proposed creating a genus Novyi and classing the species B. œdematien in this genus.

In most cases it causes in the guinea-pig nothing but edematous lesions, of a white-bluish color spreading from the point of inoculation (thigh) and covering the subcutaneous tissue of the whole abdominal membrane and even the thorax. One sees in the inoculated muscle a center of strong congestion but no gangrene.

B. œdematiens is generally very toxic, but it develops very little in the lesion created through its toxin. However there are very virulent strains, very hemolytic, capable of causing lesions exactly like those created by the V. septique. In man cases of gas gangrene are sometimes seen identical with those we have just described in guinea-pig. An instance was studied during the Crimean war by Pirogoff, who calls it "local stupor." Saquépée was among the first who observed it in the recent war; he compares it rightly to white erysipelas. Hull has also described it as a "white gangrene."

Thus you see that each of these three species most often found in the flora of gravely infected wounds, can give rise, in the guinea-pig, to the appearance of the classical syndromes, but also sometimes to quite unusual forms. That is a fact one must bear in mind to be able to understand the evolution of certain war wounds, as we shall see later on.

Finally, there is a fourth species, B. sporogenes which is sometimes able to produce a putrid gas gangrene by itself. It was seen in 1900 by Haemig and Silbermann, who wrongly described it as B. œdematien maligni. Metchnikoff and his co-workers studied it carefully.

We have been able to show that this species is quite distinct from V. septique, with which it has been often confounded. It is always pathogenic when newly isolated from the human body, and causes generally an extensive edema at the point of inoculation, which is then resorbed more or less rapidly. But sometimes very virulent strains are observed, which cause in the guinea-pig a putrid gas gangrene, quickly fatal; we saw during the war 2 such cases. In the guinea-pig there develops at the thigh a putrid myolysis which gives out an unbearable odor. We have never seen in man a gas gangrene due exclusively to B. sporogenes. However, its rôle is very important, as it is mainly its presence which causes an evolution toward a putrid form.

I must say that putridity is not, as was thought before the war, an indispensable or even very important symptom of gas gangrene; the disease can develop quite well without any fetid odor. Putridity is secondary symptom, which appears when the microbic flora contains proteolytic microbes as well as pathogenic species. In the great majority of cases the B. sporogenes is the active agent, but B. bifermentans, B. putrificus, and even a few aërobes like B. coli, B. proteus or B. pyocyaneus may have a similar action.

Exceptionally seemingly grave cases of gas gangrene are observed which are caused by B. fallax or B. aërofœtidus. Here is an example: A wounded man had had his leg cut off and a gas gangrene had developed. When I saw him a very extensive infiltration had taken place toward abdomen and thorax and the opposite thigh, the scrotum being enormously distended. As I already knew that there are anaërobes giving out an enormous quantity of gas but little pathogenic, and had seen on a rapid smear no microbe in the swollen tissue, I would not inject any serum. Gradually, the patient recovered and was well in three weeks. I was able to isolate B. aërofœtidus.

There is another very interesting microbe which plays an important part in the etiology and in the evolution of gas gangrene, without being able to produce it by itself (B. histolyticus). I have found it eight times in 91 cases of gas gangrene. A small dose injected under the skin of the guinea-pig causes a small hemorrhagic center round which the skin splits as if cut with scissors. A mortal dose in the thigh will generally kill the animal in one day and the tissues will be completely destroyed, transformed into hemorrhagic magma, and sometimes totally detached from the bone. It can

even happen that the limb itself falls off; we have seen cases when the animal has survived for a couple of days and walked about, hopping on three legs. My collaborator, M. Combiesco, has studied the histological lesions of that lysis of the tissues. He saw that the new microbe, to which we gave the name of B. histolyticus, causes an edema of a dissecting character, disrupting all the elements of the connective tissue, except only the external epithelial layer, and this, not being supported by the dermis cracks, and the internally formed magma flows through.

The same process takes place in the interior of the muscles, in the individual fibrils, whose membrane gives way as well as that of the small arteries; the blood issues and is mixed with the magma. Beckwith and Killop (1927) have shown that this microbe is capable of producing similar lesions in internal organs, such as liver, spleen, kidneys and suprarenal capsules. No other microbe is known which can produce such lesions in the animal organism.

It is quite exceptional that this microbe is found alone in the wounds. Once only I observed it, in the muscles of a left arm of a patient operated on by Dr. Broca. The skin was still intact but one could already feel, by palpation, complete lysis in the deep muscles; and in the forearm, where the wound had been, I found a diseased center where B. welchii was beginning to develop.

You can easily understand what an important part B. histolyticus can play in the etiology of a gas gangrene when it is mixed with other pathogenic anaërobes rapidly multiplying in the medium it has prepared. On the other hand, it is a very virulent microbe by itself and sometimes more toxic than the V. septique; even alone it is able to kill the patient.

To the anaërobic flora, I must add the B. œdematis sporogenes (B. sordelli), which Sordelli isolated in Buenos-Aires after the war in a case of gas gangrene. The same microbe has been also found in New York by Brewer and Melenay in a gas gangrene. In Europe it was never observed during the war. This microbe is at the same time toxic and proteolytic.

A question arises as to the grouping of the anaërobes in the wounds. In order to learn something from that standpoint, I have classified all the species found in 91 cases of gas gangrene and in 35 cases of gas phlegmon. First, and it is an important observation, every one of the 91 gas gangrene cases had been caused by anaërobes, whereas among phlegmons, 6 cases were due to aërobes. Then, gas gangrene

with one single, very pathogenic anaërobe forms about one-third of the cases (38 of 91); in the great majority of cases (29 of 36) it is the B. welchii which is the active microbe. In 26 cases (of 91), there were two microbes and in 21 there were three; thus in 47 cases there were two or three microbes; exceptionally four microbes (5 cases) were found, and five in 2 cases.

When nothing but anaërobes are taken into account, 40 different types of microbic association can be enumerated. The varieties of combination can easily explain the variation in the clinical symptoms of the gas infections.

When a missile enters the human body, it rarely introduces anaërobic microbes only; most often these latter will be mixed with aërobes, among which must be cited as the most dangerous, streptococci. In one case, we isolated seven different microbes: B. welchii, B. œdematiens, B. sporogenes; B. histolyticus, staphylococcus, streptococcus and B. proteus.

The flora of the gas gangrene is therefore essentially polymicrobic. The development of the disease depends on different factors.

1. Influence of the medium. We understand under that head, tissues, which have been compressed or crushed, flooded with edematous liquid or with blood, the missile bringing often with it pieces of cloth, and bits of earth, generally rich in chalky salts. It must be borne in mind that Bullock and Cramer have shown that small quantities of calcium salts coming from the soil favor the germination of pathogenic anaërobic spores.

2. Influence of the types of microbe introduced in the wound. As you known different strains of the same microbe display an infinite variation as to their virulence, their toxicity, their fermenting properties, etc.

3. There is also an influence in the quantity of germs introduced.

4. Finally, the microbic interaction. The evolution of the infected wound depends a great deal on the order in which the different species of the flora develop. The species which multiplies first can favor or prevent through its toxins or its ferments the development of associated microbes.

For example, my co-worker, Major Aronson, of the American staff, has shown that B. welchii, associated with a staphylococcus, gives different results according to the character of the strain of the latter, whether hemolytic or leukocidic or proteolytic. Thus, the evolution of a microbic species depends, not only on the

type of the associated species but also on the biologic properties of the strain. In order better to understand this law, one has only to study the effects of association of the B. sporogenes with different pathogenic anaërobic species.

B. sporogenes, though enhancing the development of B. welchii and being itself enhanced by this association to an equal degree, weakens greatly the proliferation of V. septique and of B. œdematiens. Thus it is far more difficult to reproduce a putrid infection with the mixture B. sporogenes plus V. septique than with an association of B. sporogenes plus B. welchii, and similarly with the mixture B. sporogenes plus B. œdematiens. When B. sporogenes is mixed with an aërobe, like B. proteus, the former may completely disappear from the inoculated region after having considerably enhanced the virulence of the associated microbe.

The factors in the evolution of the microbic flora of infected wounds are therefore very variable, and that variability explains how the wounds show such diversity of clinical syndromes.

These microbic interactions explain how it comes about that in a gas gangrene in full evolution, one sometimes finds only one or two microbic species, although the shrapnel may have introduced into the tissues a great number of microbes and of spores.

* * * *

Having described the flora and the principal factors of its evolution, we can try to establish the bacterio-clinical classification of the wound infections.

In the beginning of the war most surgeons made current use of the term "gas gangrene" to describe any serious infection of wounds, even though there was no gangrene, or gas infiltration. That was an erroneous standpoint; "gas gangrene" is not a generic term.

I thought that the term "traumatosis" was a better one to cover all the infections following a trauma; the term recalls the origin of the infection and it does not foretell anything of the clinical form which it may take. It must be completed in each case by the indication of the characteristic clinical symptom.

Thus, when the surgeon really sees a gangrene and a gas infiltration, he has to do with a gangreno-emphysematous traumatosis. If the patient does not display in the wound anything except an extensive edema, we shall describe the case as an edematous or toxic traumatosis. Some traumatoses may be simply emphysematous, or simply septicemic, when local lesions are little apparent.

But the description of the predominating clinical syndrome will not be sufficient to classify a traumatosis. We now know that the same clinical syndrome may be caused in the patient by different anaërobes. For instance, an emphysematous traumatosis may be caused by B. welchii alone, or by V. septique alone, or by B. fallax or by B. aërofœtidus, or by an association of these microbes, or by their association with other microbic species.

The final diagnosis of a traumatosis could therefore not be given before a complete bacteriological study had been made, which requires much time. Happily for the patient, we are able, as we shall see presently, to begin the treatment at once, without waiting for the particulars which the bacteriologist will observe.

* * * *

In order to fight against gas gangrene, specific sera have been prepared. As early as 1898, Leclainche and Morel, believing the disease to be caused by V. septique alone, tried to produce a serum against that anaërobe; they made use of the donkey; their serum was slightly antitoxic and of a preventive character for the guinea-pig, in the dose of 0.5 cc. against a lethal dose of purulent viscosity coming from an animal killed by the infection. Their researches had not been continued and when war broke out, we did not possess antigangrenous serum whose therapeutic value had been proved by trials on man.

As the first observations on gas gangrene did not show any pathogenic anaërobe except the B. welchii, our first serum was made against that microbe, by injecting into a horse successive increasing doses of living microbes. I take occasion here to express my thanks to the American hospital in Paris; it is there that I had to deal with the first cases of gas gangrene, and the director, Dr. Bouchet, was kind enough to offer to the Institut Pasteur a horse from which the serum antiwelchii could be prepared.

As soon as it was ready, this could be tried on man and we were happy enough to register a success in this first attempt in serotherapy. The patient, in the hospital Saint Michel, had a double fracture of the leg. Though the wound had been properly cleaned and dressed before operation, the general state of the patient became worse on the following day and a gas crepitation was noticed above the knee, at a certain distance from the wound. We then injected 22 cc. of serum antiwelchii intravenously at the elbow. That very evening, the patient got better and there were

no more gas crepitations on the following day; the wound was healthy-looking. The bacteriological examination showed only the B. welchii.

That case proved not only the efficacy of the serum, but also the value of the intravenous injection in the serum treatment of gas gangrene, as the small dose, which had been used, was shown to be sufficient to suppress the infection rapidly.

Served by chance, we encountered in the beginning of our antigangrenous serotherapy 2 other cases which were also due to B. welchii alone, at a time when we possessed only one serum, and we happily enough cured them. One was in the English hospital, the other one in the American hospital in Neuilly, in the service of Prof. Crile.

Though we were encouraged by these three first successes, more detailed researches into the flora of gas gangrene showed how illusory it was to fight all the cases of the disease without having the proper weapons, that is, without having sera prepared against all the principal pathogenic agents. Therefore we successively prepared antimicrobic monovalent or antitoxic sera, then sera which were at the same time antitoxic and antimicrobic, and finally polyvalent antitoxic sera.

As we mentioned above, the first antiwelchii serum was antimicrobic. In 1919, the American bacteriologist Bull obtained, using the horse, an antiwelchii serum which was antitoxic. In 1915, Miss Raphael and M. Frasey obtained, through a strain of V. septique from a rabbit, an antiV. septique serum which was very actively antitoxic, and at the same time Weinberg and Seguin obtained the same serum from a human strain taken in a fatal case of gas gangrene. The same bacteriologists then prepared monovalent antitoxic sera against B. œdematiens and against B. histolyticus.

We showed finally that it was possible to prepare a serum which was at the same time antitoxic and antimicrobic against each one of the five pathogenic anaërobes. The process has been described in another place. As to the polyvalent antigangrenous serum, it is a mixture of the five monovalent sera, at the same time antitoxic and antimicrobic, antiwelchii, antiV. septique, antiœdematiens, antihistolyticus and antisporogenes.

* * * *

It is easily understood that the number of recoveries in the treatment of gas gangrene had been growing at the same rate as

the number of specific sera which had been prepared, on account of the great variety of the microbic flora of the traumatosis.

After the first three recoveries in cases of B. welchii, we met with a check in an association of this anærobe with V. septique. Another very typical failure of nonspecific serotherapy of gas gangrene has been described by my collaborator Major Beebe of the American staff. It was a toxic case and as that form is generally caused by B. œdematiens, he injected, at first sight, the specific serum against that microbe. The patient died; the bacteriological examination, made by both of us, showed a very rare instance of an edematous traumatosis caused by B. welchii.

It is useless to give more examples of such failures, which are cited in a book of ours.

An appreciable improvement was introduced into the serotherapy of gas gangrene as soon as we were able to inject into our patients a mixture of three sera (antiwelchii, anti V. septique and anti-œdematiens). The results of this method, which could be communicated by Weinberg and Seguin to the French academy of sciences in July, 1917, showed 24 cases of gas gangrene with 19 recoveries.

Our researches, continued during the year 1918, gave a still better percentage of cures. The cases numbered 60, 42 of which were treated by a mixture of the three sera, and a fourth one, anti-histolyticus, was added for the 18 other patients. We had altogether 10 failures. In one case, we are entitled to say that the patient succumbed to an accident apart from gas gangrene. He had been wounded in the thigh and the infection had spread over buttock and abdomen. The serum having been injected, the limb regained the proper aspect and the general condition became normal after a few days, but death was caused by a hemorrhage of the femoral artery on the tenth day due to injury from an adjacent fragment of shell.

Two other cases were due to V. septique. The serum completely cured the patients as regards that microörganism but they died from a septicemia caused by a streptococcus. In a fourth case, the patient died from an infection of B. histolyticus, for which an antiserum was not yet included in the mixture. These 4 failures can therefore not be put to the debit of serotherapy.

There are still 6 failures to be accounted for. Two patients were already in their death agony when we came to their bedsides and they died while being injected; two more died one hour after injec-

tion; the fifth one four hours later and the sixth twelve hours after the injection. It can therefore be said that in the whole 60 cases, the serum treatment has failed only in the last four.

If we consider especially the 18 more recent cases, just before the armistice (16 in the service of M. Chutro and 2 in that of M. Abadie) in the hospital of rue de la Glacière, the results are really remarkable. We had only 1 failure, the case mentioned above as, due to B. histolyticus. Taught by experience, we gave the 17 other patients an injection of four sera mixed together, and all recovered. It is especially noteworthy that these patients had all been chosen from among the most serious cases of gas gangrene, in order to see the specific serotherapeutic value of our serum. In 2 of these cases, the patients displayed in the wounded limb not only gas infiltration, edema, bronzed patches, but also numerous hemorrhagic phlyctenes, a symptom necessitating a hasty amputation, besides, that symptom signifies always a fatal issue if the amputation cannot be performed.

Study of our last series of cases shows clearly what appeared probable from the researches on the microbic flora of gas gangrene, namely, that the serotherapeutic treatment of the disease by the injection of a polyvalent serum (mixture of four specific antisera) offers the highest chance of recovery. When the wound gives out a putrid odor, it is useful to add also a certain quantity of antisporogenes serum; otherwise it is not necessary as the normal serum of the horse contains a sufficient quantity of sporogenes antibodies to help the organism to destroy the few elements of that species which might be among the microbic flora of the wound.

These last observations also again showed the excellence of the intravenous method of introducing the antigangrenous serum, as we had seen from the very beginning of our serotherapeutic studies.

Effectively to cure gas gangrene, it is necessary to bring about as rapidly as possible the neutralization of the toxin or the toxins already in the organism, phagocytosis of the pathogenic germs which have come into the blood, and also local destruction of toxin and phagocytosis in the wound. When the wound has been properly dressed, this general result can be very rapidly obtained through a single intravenous injection of the quadrivalent serum.

When a patient is treated intravenously and when the wound has been well cleaned out by the surgeon, after débridement, it is not absolutely necessary to apply the serum locally. However, the local application of the serum by injection into the wound

or by direct application on the dressing, will contribute to rapid convalescence.

Our therapeutic successes were confirmed by a great number of surgeons in Paris and in the hospitals at the front. The more important observations have been published by Prof. Delbet; Prof. Duval; Dr. Vaucher and Prof. Chutro.

Our experiments have also revealed that the polyvalent serum possesses a high preventive value. At our suggestion, our friend Dr. Vaucher made a first experiment in preventive serotherapy at the front and it was very successful. Then, with Dr. Duval, he made a second experiment on a larger scale. They were able to show that a mixture of antigangrenous sera lowers considerably the percentage of cases of gas gangrene among very seriously wounded soldiers; it was shown also that the injection into the muscles gives better results than the subcutaneous injection.

The number of registered recoveries obtained by antigangrene serum is very great. The French and Spanish have used the serum in the recent Moroccan war. In current practice, the serum is very helpful in the treatment of gas phlegmon, whatever the diseased organ may be: in anal fissures, cancer of the rectum, and of the neck or the body of uterus, and also in cases of osteomyelitis (Mastier 1923; Weinberg and Davenne, 1928). It has given favorable results in gangrenous infections of the most varying types, as noma (Débérnidetti and Geslin, 1927), in Ludwig's angina (Gamaleua and Mathieu, 1923), Hallopeau, Lheureux, 1924), in gangrenous angina with tonsillar phlegmon (Tixier, 1923; Rouffiac, 1930), in the gangrene of the pharynx (de Massary and Boulin, 1921), in gangrenous sinusitis (Verger, 1929), in the phlegmon of the thoracic membrane (Rouffiac, 1930), in gangrene of the penis and of the scrotum (Chauffard, Zuber and Marguezy, 1922); (Lortet, Jacob and Bureau, 1929), in gangrenous vulvovaginitis (Guy, Laroche and Deglaire, 1922). Finally, there are some cases of recovery in diabetic gangrene (Chauffard, Zuber and Marguezy, Lorente d'Amposta, 1921; Spreafico d'Archena, 1923).

Before concluding, I feel it necessary to remind you of some practical indications. It must not be forgotten that the serum treatment of gas gangrene is only complementary to the surgical intervention, which is indispensable. The surgeon must open the wound, extract every foreign body, cut out all the damaged tissues and injured arteries; otherwise, the serum cannot penetrate where it will be useful.

If the gas gangrene is at the beginning of its evolution, one must inject under the skin of the patient 100 cc. of polyvalent antigangrenous serum, diluted in 400 cc. of physiological water. If the operation can be performed at once, it is better to make the injection just at the end, while the patient is still under the anesthetic; in that way every anaphylactic phenomenon can be avoided. When the case is very serious, when the general condition of the patient is bad, the pulse feeble, one must not hesitate to make at once an intravenous injection of the same amount (100 cc.) diluted in 1000 cc. of water.

When the patient shows that he feels unwell during the intravenous injection, which must be made at a very slow rate, by congestion of the face, shivering, and dyspnea, it is better to stop the injection and to resume when the patient feels better. Sometimes, half an hour to two hours after the injection, the patient may feel a sudden shivering, which may last for an hour. After the shivering, the temperature may rise to 40° C. (104° F.). On the following day, the temperature will be lower and the general condition improved. The edema of the limb will begin to subside, the dressing will acquire a yellow coloring, due to an abundant flow of lymph. Sometimes the injection must be repeated on two or three successive days.

By employing this technique, it is possible to fight with success the classical gas gangrene and equally well all infectious wound complications caused by anaërobes, whatever may be the clinical syndrome they display. It is effectively proved by the bacteriological researches made during the war that the anaërobic flora of infected nongangrenous wounds never contain any other pathogenic anaërobes than those which have been isolated in gas gangrene.

Moreover, according to our observations, very serious wounds treated by antigangrenous serum have never shown any complication due to staphylococcus, though it is often to be found in the wound; on the other hand, we have seen wounded persons who had been freed from gas gangrene die of infection by streptococcus. The fact is easy to explain: normal horse serum contains antibodies which are active against staphylococcus but are generally completely inactive against streptococcus. When the microbic flora contains streptococci, it is therefore useful to add some antistreptococcus serum to the antigangrenous serum.

Finally, I must add that it is always necessary to treat prophylac-

tically by the administration of a mixture of antitetanus and antigangrenous serum those injured in accidents, since these wounds are contaminated not only by B. tetanus but also by anaërobic gas gangrene microörganisms. Moreover, we know that the presence of the latter facilitates the development of B. tetanus.

You see then, my dear colleagues, that the researches made during the war on the infection of wounds have been very fruitful. The microbic flora of traumatoses has been well studied in detail; thanks to that study, it has been possible to establish a logical classification of the different clinical forms of the wound infections (or of traumatoses) and to show that the classical gas gangrene is only a putrid variety of the gangreno-emphysematous form of traumatosis. Moreover, we were able to establish the technique of serotherapy of gas gangrene and, in general, of all the wound infections caused by anaërobes.

To conclude, I must say that our hopes have been more than fulfilled, as the results acquired in one direction have incited bacteriologists to extend their studies on the rôle of anaërobes in other polymicrobic infections, such as appendicitis, peritonitis, pulmonary gangrene, puerperal infections, complications of typhoid fever and other intestinal infections. But I must stop; I will only say that these researches have already yielded very interesting and promising results.

THE MANAGEMENT OF GOITER AND HYPERTHYROIDISM*

By FRANK H. LAHEY, M.D.
BOSTON, MASS.

THE MÜTTER LECTURE

One cannot receive the invitation to give the Mütter Lecture without being very conscious of the honor and obligation which go with it. As to the honor, I am completely sensible of its degree and appreciative of it. The obligation, as it appears to me, is not in the giving of the lecture, but that the lecture must present to you the results of a personal experience and personal deductions together with those associated deductions made by intimately related colleagues working in a closely linked group.

The remarks which I will make, then, will present to you something of a clinical summary of our experiences and conclusions in the various thyroid states, based upon an operative experience in the clinic now of over 9000 operated goiter patients, and, of course, a nonoperative experience, also, with a further quite large group. It will be my purpose to speak particularly of the points which I think may be of value to you as physicians directly handling these conditions in patients, rather than to direct my remarks specifically toward those doctors who are handling these patients surgically.

The first condition concerning which I desire to speak is the so-called diffuse colloid goiter. This goiter is the soft, symmetrical enlargement which so frequently appears in young girls and young men, unassociated with symptoms of toxicity. It is the type of goiter which appears so commonly in the form of what is called adolescent goiter. It is also the type of thyroid enlargement which first appears as a forerunner of the nodular endemic goiter which occurs in regions where goiter is prevalent and where there is usually an iodine deficiency in the drinking water, there being less than 1 part of iodine to 1,000,000,000 of water.

* Read December 3, 1930.

Frequently the first evidence of goiter in thyroid enlargement of this type is a visible isthmus which may be seen to ascend and descend as the patient swallows. It is because of the presence of this visible isthmus that many of these patients are sent to us for an opinion as to whether or not they are causing nervousness and tachycardia, symptoms which are often present in young girls. In this connection one should realize that very few of these diffuse colloid goiters in young people have real associated toxicity, that young girls and boys at this age, with particularly sensitive nervous mechanisms (general and cardiac), are very susceptible to emotional and nervous stresses. It is in this group of cases that critical clinical and laboratory investigations must be done and sound clinical judgment maintained. It is very easy to associate the presence of thyroid enlargement with a tachycardia and nervous symptoms, and to consider them as of thyroid origin, when in many instances they are not of thyroid, but of neurologic origin, and the thyroid enlargement is merely coincident and actually unrelated to the chain of nervous symptoms. It is in these cases that careful clinical histories should be obtained and repeated basal metabolic tests done.

On should realize that real hyperthyroidism is usually associated with thyroid hyperplasia and that thyroid hyperplasia usually causes definite firmness in the glands. When, therefore, the symmetrical enlargement of the thyroid is soft in character, particularly when occurring in a young person, considerable suspicion should exist that associated nervous symptoms are not of truly thyroid origin. A great many young girls of this type have, we think, been needlessly subjected to partial thyroidectomy when more careful investigation would have demonstrated the absence of any real hyperthyroidism.

Diffuse colloid goiter occasionally does demand surgical removal. Occasionally goiters of this type attain such size that they are quite unsightly, and so justify their removal to improve the appearance of the patient. Occasionally diffuse colloid goiters become toxic after existing for months or years without toxicity, and demand removal for such a reason. Occasionally diffuse colloid goiters produce sufficient pressure upon the larynx so that it becomes necessary to remove them for this reason. Tracheal obstruction due to goiter in practically all instances except in the case of diffuse colloid goiter is secondary to intrathoracic extension of the goiter

deviation, counterpressure and narrowing of the trachea. Almost never does diffuse colloid goiter become intrathoracic. This is due to the fact that it takes time for a goiter to grow sufficiently to extend down into the thorax. Diffuse colloid goiter is the preliminary stage of nodular, multiple, colloid adenomatous goiter, the type of goiter which one sees in people who have had goiter for some time and in a region where goiter is endemic. When, therefore, a diffuse colloid goiter has existed long enough to extend downward into the thorax, it has usually existed long enough to have degenerated from a diffuse colloid goiter into a nodular multiple colloid adenomatous goiter.

Diffuse colloid goiter does occasionally produce interference with breathing, but it is most often due to the progressively enlarging upper poles of the diffuse colloid goiter extending behind the larynx in the form of retrolaryngeal extensions and producing pressure on the soft, unprotected, posterior wall of the laryngeal cartilage.

We believe, then, that diffuse colloid goiter in young girls is usually not associated with toxic symptoms, and that surgery is indicated in goiters of this type for unsightliness, toxicity when it can be demonstrated, and because of respiratory obstruction secondary to thyroid pressure.

The next thyroid state of which I wish to speak is adenoma of the thyroid. In any discussion of adenomata of the thyroid, it is essential that a clear definition of what are termed adenomata be made, in order that confusion may not arise between the degenerative type of nodular goiter, so commonly seen in regions where goiter is prevalent, and the true tumor of the thyroid, seen in our community so frequently as a single discrete thyroid tumor occurring in a thyroid which, exclusive of the portion occupied by the tumor, is otherwise normal.

The true adenoma of the thyroid, called fetal or embryonal adenoma of the thyroid, is seen by us most frequently as a single (although it may be multiple) encapsulated tumor involving either lobe or the isthmus, and varying in size from the size of a grape up to the size of a large football. They have thick and definite fibrous capsules, and contain tissue varying from completely undifferentiated thyroid tissue up to completely differentiated thyroid tissue. The tissue within these adenomata varies from firm vascular normal thyroid tissue to edematous unvascular tissue, with degrees of tissue necrosis or degeneration up to complete lique-

fication and cyst formation. Exclusive of an occasional rare cyst of the thyroid, due to hemorrhage, practically all cysts of the thyroid are represented by adenomata of the thyroid whose centers have become necrotic, the resulting liquid being retained by the true fibrous capsule of the adenoma, thus producing a cyst.

It is probable that these discrete adenomata are of true rest origin, a deduction which is supported by their proneness in our experience to be single, and the fact that practically all cancer of the thyroid (96 per cent in our series) arises in tumors of the thyroid of this type. One should realize that the so-called multiple colloid adenomatous goiter (so often endemic in type), characterized by multiple nodular adenoma-like tumors, is really not composed of true tumors, is not susceptible to malignant degeneration as is the true adenoma and in all probability is the result of a degenerative process and not a new growth. The nodules in endemic or multiple colloid adenomatous goiters do not possess true capsules. They do not contain undifferentiated thyroid tissue. They do not contain healthy vascular thyroid tissue, but necrotic colloid-laden, degenerated, thyroid tissue. In our discussion, therefore, of adenomata of the thyroid we wish to exclude from consideration under the head of adenomata this nontumor type of nodular pseudotumor goiter, and include only those embryonal or fetal adenomata spoken of above under the head of true encapsulated thyroid tumors.

Practically all thyroid clinics which have investigated the incidence of thyroid malignancy have found that of all thyroid malignancy, 90+ per cent have originated in previously existing benign fetal adenomata.

If the criteria of evidence of malignancy in thyroid tumors be limited solely to the morphologic features which characterize malignancy elsewhere, then the percentage of malignant degeneration in our experience is about 2 per cent. We know, however, that, although a tumor of the thyroid may retain morphologic features of a benign character, still the tumor may metastasize and even the metastatic growth may still lack the typical microscopic appearances of malignancy. This feature in adenomata of the thyroid explains the group of adenomata which continental pathologists in the past termed malignant adenomata because of their ability to metastasize while still adenomata. We are indebted to Dr. Allen Graham for elucidating some of the hitherto unappreciated features of adenomata. Dr. Graham has shown that in

certain adenomata a rupture of the wall of the bloodvessel occurs in some parts of the adenoma with ingrowth of the adenomatous tissue into the vascular canal, a demonstration which may explain the ability of some of these tumors to metastasize without typical malignant microscopic features. Dr. Graham has called attention also to the fact that metastases from adenomata of the thyroid which have become malignant, but have not ruptured the capsule of the adenoma, tend not to metastasize into the adjacent lymph nodes, but to metastasize by the bloodstream.

In a series of 1484 discrete fetal adenomata of the thyroid studied in this clinic 92, or 6 per cent, showed either frank or potential evidence of malignancy. For that reason, then, and because operations for discrete adenomata of the thyroid are attended by a very low mortality, we have taken the position that all discrete adenomata of the thyroid in women aged thirty years on shall be removed in order to obviate later danger of malignancy in them.

Adenomata of the thyroid not infrequently attain such size that they are quite disfiguring, and when they do so we believe that this is a sufficient indication for surgery to justify its recommendation.

Adenomata of the thyroid not infrequently, when they originate in the lower poles of the thyroid, slip downward behind the clavicle and sternum, into the superior mediastinum, where they become intrathoracic in location. We believe that any goiter which is intrathoracic, or located so low in the neck behind the clavicle or sternum that it is tending to become intrathoracic, should be removed for these reasons.

Intrathoracic goiter is almost always associated with some degree of lateral tracheal displacement and narrowing of the caliber of that structure. Not infrequently, therefore, the patient's attention is first directed toward such a lesion by noisy breathing (respiratory stridor) or by interference with breathing. Occasionally, as a result of the narrowing of the trachea from the pressure of intrathoracic goiter on the trachea, there are periodic attacks of serious suffocation, occurring usually at night. These, we believe, are due to the accumulation during sleep of thick and tenacious mucus in the trachea below the narrowed portion of the trachea which, when sufficient mucus accumulates, occludes the lumen at its narrow part and wakes the patients up in a state of serious panic and causes them to struggle violently for their breath. As

the result of their fearstricken and powerful exertions they succeed in dislodging the mucus through the narrowed portion, with immediate relief and restoration of normal breathing. One should, therefore, suspect intrathoracic goiter with tracheal narrowing in any patient with these unexplained attacks of respiratory difficulty.

Intrathoracic goiters not infrequently so completely fill the upper thoracic strait that they encroach upon the caliber of the internal jugular veins, lessening thus the capacity of those two main venous channels to take care of the return blood supply from the head and neck. In such cases the venous return is augmented by a compensatory dilatation of the superficial thoracic veins. This causes dilatation of the veins on the anterior chest wall, and, therefore, one whould be suspicious of intrathoracic goiter in any patient showing dilatation of the superficial thoracic veins on the anterior chest wall.

Intrathoracic goiter occasionally shows dulness on percussion over the upper chest, but their presence is much more reliably demonstrated by *x*-ray pictures.

Since most intrathoracic goiters are of the adenomatous type, they usually originate either in the right or left lobe, and so are located lateral to the trachea in their intrathoracic situation, and tend to deviate that structure to one side in a typical curve corresponding to the curve of the adenoma itself. The intrathoracic extensions of the thyroid show typical shadows in the upper mediastinum, extending down to and often well below the arch of the aorta. Since many of the intrathoracic goiters are discrete fetal adenomata, and so possess distinct, thick, fibrous capsules, the outlines of the intrathoracic shadows are the sharp and clearly defined curved outlines of the adenomata.

We believe, therefore, that all patients of thirty years or over with discrete fetal adenomata of the thyroid should have them removed to avoid that later danger of malignancy in them. We believe that they should be removed when they are of sufficient size to be unsightly. We believe they should be removed when they are associated with toxicity and when they tend to or have become intrathoracic in location.

I would like, now, to discuss the subject of toxic goiter. One hears and reads considerable regarding the division of toxic goiter into two types—the primary hyperthyroidism or true exophthalmic goiter and the secondary hyperthyroidism or so-called toxic adeno-

mata. We believe that there is no physiologic evidence to prove that these are different diseases, and from the point of view of simplicity we think it is desirable to consider and discuss hyperthyroidism all under one head It must be admitted that although there are certain minor differences between true exophthalmic goiter and hyperthyroidism associated with adenomata, such as a tendency for the basal metabolism to be higher in exophthalmic goiter, and for the disease to be less intense and active in character in toxic adenomata, nevertheless, they are basically so similar and react so similarly to operative procedures as relates to clinical improvement and the return of basal metabolism to normal levels, that we think they should be grouped under one and the same head.

The eye signs of exophthalmic goiter as described in the various textbooks—such as the inability of the eyes to converge upon a near point equally, known as the sign of Moebius; the lid lag, known as the sign of von Graefe; the widening of the palpebral fissure and the diminished frequency of winking—are deserving of less attention than they have received in textbooks in the past. We know that all of these signs may be absent and still hyperthyroidism be present. We know that exophthalmos is at times absent and still hyperthyroidism present. We know that when these signs are present, rarely are more obvious and definitely convincing clinical signs lacking. The eye sign which is quite reliable in all toxic goiters is stare. Rarely does hyperthyroidism exist in the absence of some fixation of the eyes. Experience with hyperthyroidism leads one to distinguish this stare quite readily, and it varies in its degree in quite definite relation to the degree of intoxication.

One should not discuss the eye signs of exophthalmic goiter without calling attention to the danger of blindness in any patients with marked exophthalmos. In our experience with this disease we have seen six eyes lost due to slight erosions of the cornea, followed by ulcerative edema and sloughing, which required removal of the eye.

One should take every precautionary measure against any possible injury to the cornea in these patients with marked exophthalmos, and when redness, conjunctivitis or ulceration has occurred positive measures must immediately be undertaken unless one wishes to see first one eye and then the other become edematous, infected and require enucleation. Suture of the lids, with denuda-

tion of lid edges, will be necessary in many of these cases, and in spite of this in our experience the process will sometimes not be stopped.

The most important point in connection with marked exophthalmos is to consider it an indication for early subtotal thyroidectomy, and so bring about diminution in the exophthalmos before the above complication has arisen. We believe that the elimination of prolonged conservative treatment and the earlier application of surgery in those patients in whom we have seen the eyes lost would have prevented this depressing situation.

It should be realized in toxic goiter that the size of the thyroid plays little or no part in the degree of intoxication. Not infrequently one sees very large, firm, exophthalmic goiters with relatively low degrees of thyroid intoxication. On the other hand, one not infrequently sees thyroids not only of normal size but of little less than normal size, but firm and hyperplastic, with extreme degrees of thyroid intoxication, and it is due to the fact that exophthalmos and goiter are frequently absent that the early diagnosis of this condition is sometimes overlooked.

Tachycardia is a very dependable clinical feature in hyperthyroidism. Roughly, the degree of tachycardia approximates the degree of intoxication. One should bear in mind, however, that it is possible to overlook tachycardia, particularly in the patient who is in bed at rest and has been so for a considerable period of time. A pulse-rate under these conditions may be deceivingly low and so not attract attention to unsuspected hyperthyroidism. It is well, therefore, in all patients in whom hyperthyroidism is not frankly evident, but is suspected, that the pulse-rate be taken with the patient up and about.

The clinical history in hyperthyroidism is of a great deal of importance. Many of these patients give histories of unexplained periods of nervousness, unexplained periods in which weight is lost in spite of a good appetite. The history of weight loss in hyperthyroidism should always be correlated with the question of food intake. It is dependable as a clinical feature when weight loss is definitely associated with no variation in the food intake or occurs in spite of an excessive intake. Changes in disposition in the form of irritability, as noted by the family and friends, are suggestive clinical features. Change in the ability of the patient to endure amounts of activity which were previously well endured suggests

the myasthenia which so frequently accompanies hyperthyroidism. Changes in menstrual flow are also of value, the most common change being that of diminution, but occasionally we have noted excessive flowing.

One cannot, of course, discuss the question of hyperthyroidism without the consideration of basal metabolism estimations. There is no disease with which we deal in which the basal metabolism is elevated so characteristically in relation to the clinical symptoms as in the case of hyperthyroidism. Elevation of the basal metabolism occurs in other conditions, such as the leukemias, some cases of pernicious anemia, certain cases of cardiorenal disease and in diseases with increased muscular tone, as in Parkinson's disease, but its most typical elevation occurs with hyperthyroidism.

It is but natural that, since basal metabolism is an indication in percentage figures of the degree of intoxication, considerable error should arise in connection with its use. One should, I think, always realize that basal metabolism in hyperthyroidism is of value only when it is correlated with clinical findings. One should realize that were the diagnosis of hyperthyroidism made primarily on basal metabolism readings, there would be a very high percentage of error. It should be realized that when the basal metabolism is characteristically elevated, such as to +35, +40 and +50 or over, there should be present very obvious clinical symptoms of hyperthyroidism, such as a firm gland, usually eye signs, particularly stare, tachycardia, weight loss, nervousness and tremor, to correlate these basal rates if thyroidism is truly present in a degree to elevate the metabolism to this level. When basal metabolism is elevated to such degrees as +50 and over, and obvious clinical signs of hyperthyroidism are not present, one should view such reports with suspicion, ask for a repetition, and again investigate the patient clinically for the possibility that his symptoms are not of thyroid origin and that +50 is not his true basal rate.

In general, as relates to basal metabolism rates, when + rates are as high as 50 or over, clinical symptoms of a distinguishing character should be present. In the same way, when basal rates are strikingly low, −25 or lower, rarely will the distinguishing clinical features of myxedema be absent. Many patients have − basal rates up to −20 to −25 without the obvious signs of myxedema, but, as stated above, from −25 downward this is not so.

As relates to the diagnosis of myxedema in patients with − basal

rates between −10 and −25, assistance may be obtained from the fact that the blood cholesterol has been demonstrated by Dr. L. W. Hurxthal and Dr. R. L. Mason in this clinic* to be elevated in those patients with definite myxedema, and normal in those patients with − rates but without myxedema.

We feel very strongly, then, that basal-metabolism estimations, to be of value in hyperthyroidism, should be carefully safeguarded (1) as to the accuracy of the test, and (2) to its correlation with clinical symptoms.

In order that the test may be done with the greatest degree of accuracy, we have insisted that patients come to the hospital and stay there the night before, so that we may be certain that factors do not enter into the test during this period which may elevate the rate and thus make it an untrue test. Errors, also, as relate to the test, occur not infrequently with the apparatus. Unless the metabolism apparatus, particularly of the closed circuit variety, is being employed constantly, leakage in the apparatus or channeling of the soda lime will occur which will result in tests which are not true estimations of the basal metabolism. We feel that every basal metabolism apparatus should be checked against a normal individual at least several times a week and preferably once a day, and that one apparatus should be frequently checked against another apparatus to insure its accuracy.

When basal metabolism readings are repeated and vary, either at the same test or from day to day, one should suspect that they are not true tests, and that they possess a considerable possibility of error. For example, when a patient is submitted to three periods, the first of which is +25 and the second +65, it is obvious that some error exists in either one of the tests and measures should be undertaken to correct this. It is, also, evident that when a patient has a metabolism of +75 or +80, and yet lacks the clinical evidence of such a degree of intoxication, caution should be employed in arriving at a diagnosis of hyperthyroidism and the test should be repeated, to make certain that the rate is not unduly elevated for causes other than those of thyroid origin.

It has been our experience in dealing with a great many thousand basal metabolism tests that actual active hyperthyroidism does not

* For complete data concerning this observation, see paper recently sent to the New England Journal of Medicine by Drs. Hurxthal and Mason, of this clinic, and Miss Hazel Hunt, of the New England Deaconess' Hospital.

exist in the absence of an elevated basal metabolism test, and, likewise, that when a patient has actual hyperthyroidism, with an elevation of his basal metabolism, he is not cured of that hyperthyroidism until his basal metabolism has reached a normal level and has maintained a normal level for a considerable period of time. We believe that any patient who can be said to have been cured of hyperthyroidism must show a normal basal metabolism at least one year after his operative or treatment procedure.

There are many patients with hyperthyroidism of the borderline character in whom the diagnosis of hyperthyroidism is extremely difficult and, from clinical findings, impossible. In such cases we find that it is of great value to send these patients in the hospital to do metabolism tests upon them every day for several days, in order that not a single basal metabolism may be reported, but that the trend of basal metabolism may be reported. If these patients' symptoms are of truly thyroid origin, then, even though the test be repeated daily for several days, it will still show persistently elevated rates. If the symptoms are not of thyroid origin, but are of neurotic origin, then as the patient becomes adjusted to the tests, the first test, which will be elevated, will gradually be lowered each day as the patient loses his apprehension for the test and adjusts himself to the procedure, until eventually it will reach or be close to normal. It is, therefore, wise in borderline cases not to depend upon a single basal metabolism reading, but to submit the patient to several tests over several days and establish whether the trend of the test is to remain elevated or to approach normal.

We believe it is a great mistake to make a diagnosis of hyperthyroidism in the absence of an elevated basal test. It is possible during a period of remission that the residual symptoms of hyperthyroidism, such as nervousness and tachycardia on exertion, and exophthalmos may still be present, and so make it possible to arrive at a clinical diagnosis of hyperthyroidism. One can never be sure that this period of remission as demonstrated by a normal basal metabolism may not be prolonged into a permanent one, and we consider it unwise and unjustifiable to operate upon patients during this period if their basal metabolism is normal. It is better, we think, to wait and observe such patients, and if their metabolism rises again, then to submit them to surgery.

Due to the prevalence of the employment of iodine, borderline cases of hyperthyroidism are often masked by having had iodine

over a considerable period of time before coming to us for an opinion. In such cases we frequently find it necessary to send the patients home for three or four weeks, taking them off iodine, and to have them return for another basal metabolism and clinical examination at the end of this period.

Likewise, it has proven of value in doubtful cases at times to put patients on iodine in the form of Lugol's solution for approximately two weeks, to see whether or not it would produce any drop in basal metabolism or clinical improvement, providing thus an added clinical feature in favor of hyperthyroidism in cases otherwise not diagnosable.

Apathetic Thyroidism. Everyone who practises medicine is entirely familiar with the young girl with typical activated hyperthyroidism. Not everyone, however, is familiar with the fact that there exists another type of hyperthyroidism which we have termed apathetic hyperthyroidism, quite different in character from that of the activated type.

We have discussed and written about apathetic thyroidism now for several years, but I feel that it is still often not recognized, with the result that not infrequently these cases exist undiagnosed over long periods of time and so are often submitted to surgery at a very late date. Comparison of some of the clinical features of the two types will be of service in fixing in your minds the differentiation of the two kinds of thyroidism.

In exophthalmic goiter of the active type exophthalmos is frequently present. In apathetic thyroidism, exophthalmos is usually not present, and even stare is not of a marked character. In activated hyperthyroidism in the young person, for instance, the thyroid is usually enlarged and firm in character. In apathetic hyperthyroidism the thyroid is frequently not enlarged, is quite small, but is firm and frequently of pebbly character. Activated hyperthyroidism usually occurs in people aged forty years or under, while apathetic hyperthyroidism occurs in more elderly people aged forty years or over. The apex-beat in activated hyperthyroidism is forceful and of the tumultuous and thrusting character, often being diffused over the entire precordia. The apex-beat of apathetic hyperthyroidism is very much less forceful, is not distributed, as a rule, over the precordia and is not impressive or thrusting in character. The character of the pulse in hyperthyroidism is full, snapping and typically toxic, while the pulse in apathetic hyperthyroidism is very much less striking, is not toxic and snapping in

character. The pulse-rate in activated hyperthyroidism tends to be high, varying with the slightest activation, while the pulse-rate in apathetic hyperthyroidism is frequently relatively low, 120 or under. The skin in activated hyperthyroidism, particularly in young individuals, is hot, moist and flushed, while the skin in apathetic hyperthyroidism is dry, cool, pigmented and more or less of senile character. The appearance of the individual with exophthalmic goiter of the activated type is one of youthfulness, due to the fact that it occurs in younger individuals and also due to the fact that skin moisture, skin heat and the flushing tends to present in the individual a youthful appearance, while the appearance of the patient with apathetic hyperthyroidism is one of senile character, many of the patients appearing ten years or more older than their actual age. This appearance of senility is, of course, induced by the pigmentation, by the marked loss of weight which frequently is present in the apathetic hyperthyroidism, and by the dry character of the skin. Weight loss in activated hyperthyroidism is of considerable amount, but varying in character, not infrequently existing for a few weeks and then being regained, while the weight loss in apathetic hyperthyroidism, although not striking, is usually considerable and progressive in character. The basal metabolism in activated hyperthyroidism tends to be high and in proportion to the intensity of the symptoms, while the basal metabolism in apathetic hyperthyroidism tends to be relatively low, +35 and under. The whole picture of apathetic thyroidism is not a striking one, and is one which often readily leads either to the diagnosis being overlooked or the severity of the intoxication being underestimated.

Young patients with activated thyroidism die in a typical way. We have seen a number of young individuals with activated hyperthyroidism die characteristically in excitation, with uncountable pulses, with marked delirium, with extreme activation and with all the signs of intense intoxication. On the other hand, when patients with apathetic hyperthyroidism die the picture is quite reversed. They sink gradually into unconsciousness and coma and die without activation, without delirium and without striking tachycardia.

It is because of the unrecognized seriousness of apathetic hyperthyroidism that many elderly patients with this disease are wrongly considered good risks and succumb to unjustifiable complete one-stage surgical procedures on them.

It should be realized that every patient with apathetic hyperthyroidism, no matter how mild his symptoms may be, possesses the potential possibility of coma and postoperative death. So strongly do we feel concerning this at the present time, that when we feel sure that a patient with hyperthyroidism is of the apathetic type, we seriously consider a two-stage operation, no matter how well the patient may run his course as relates to pulse-rate and pulse-pressure on the operation table. We know that the patient with apathetic thyroidism possesses always unknown and inestimable possibilities of untoward postoperative reactions. Even though his pulse during subtotal thyroidectomy may never exceed 120, even though his preoperative basal metabolism may not be over +30, even though preoperatively he may have been assumed to be a good surgical risk still he may return to his bed, never recover consciousness from anesthesia and die in the typical coma of apathetic thyroidism. It is because of the possibility of the unexpected fatalities in apathetic thyroidism that we stress its existence as a type of thyroidism that demands careful and conservative management if fatalities are to be avoided.

Thyroid Crises. When iodine was first introduced we hoped that we would no longer have to deal with patients in thyroid crises; that is, the patients who come to us or whom we are asked to see in delirium, with vomiting and diarrhea. We hoped that the employment of iodine would bring these patients out of crises and permit us to eliminate from our experience the mortality associated with crisis. Unfortunately this has not been so.

It has been our observation that when patients are permitted to get into real thyroid crises, a majority will die in spite of all corrective measures. We feel very strongly that thyroid crises represent an emergency which should be met very similarly to diabetic coma. Patients can be dealt with satisfactorily when the crises are impending, but not when they have developed. If recognized during the impending stage of the condition a great majority can be kept out of crises and will not die. If, on the other hand, these measures are not undertaken until patients have entered crises, *i. e.*, delirium and exaggerated activation, then, as stated before, a number of these patients will die in spite of all measures to prevent them.

We feel, therefore, that when any patients with severe hyperthyroidism show impressive signs of the rapid intensification of

the toxicity, such as persistent increases in pulse-rate, progressive intensification of nervous symptoms, marked increase in weight loss, vomiting and diarrhea, any change in the mental state, such as wild or irrational statements, one should be suspicious that the patient is on the verge of a thyroid crisis. Under such conditions one should devote his attention toward measures to prevent the further intensification of this toxicity. This can best be accomplished by the immediate administration of at least 2500 cc. of salt solution daily, with at least 75 gm. of glucose, the administration of sufficient morphine to control the patient's restlessness (patients with active hyperthyroidism tolerate large doses of morphine) and the administration of iodine in the form of Lugol's solution by mouth, by stomach tube, by rectum or in the hypodermoclysis, 50 minims in 1000 cc. of salt solution. It has been our experience that when these emergency measures are undertaken early enough, patients can be kept out of crises, gotten into good condition and operated upon within two to three weeks by first-stage subtotal hemithyroidectomy. Their toxicity is thus cut down, and the danger of reëntry into the crisis is overcome. At the end of six weeks the second-stage left subtotal hemithyroidectomy can be done safely and the thyroidism permanently checked.

Diabetes and Thyroidism. As relates to hyperthyroidism and diabetes, Dr. E. P. Joslin and I have reported in the *American Journal of the Medical Sciences* (1928, clxxvi, 1) our experience with 75 such cases. A combination of diabetes and hyperthyroidism is a very undesirable one from the point of view of control of diabetes. It is very similar to the association of diabetes and infection. It is impossible to satisfactorily manage many of these patients as to their diet and insulin until their hyperthyroidism is controlled.

The increase of glycosuria in hyperthyroidism is so definite that one cannot escape the deduction that it is the result of the hyperthyroidism. Thus in a series of 228 cases of primary hyperthyroidism 38.6 per cent showed 0.1 per cent or more sugar in their urine before operation, or 0.5 per cent or more after operation; while in 83 cases of adenomatous goiter with secondary hyperthyroidism 27.7 per cent showed the same per cent of glycosuria present. The significance of these figures can be appreciated when it is realized that in 189 cases of nontoxic goiter this percentage of glycosuria was present in only 14.8 per cent of the cases. This 14.8 per cent of patients showing glycosuria corresponds with the

per cent of glycosuria occurring in 500 successive surgical cases entering the New England Deaconess Hospital not having either diabetes or hyperthyroidism. Even among a series of 100 patients with disease of the gall-bladder, not having diabetes or hyperthyroidism, the incidence of glycosuria was only 16 per cent.

So definite is the relation of glycosuria to hyperthyroidism that it has been necessary for Dr. Joslin to raise the standard for diagnosis of diabetes in hyperthyroidism from above 0.13 per cent fasting or 0.16 per cent after a meal and with glycosuria to 0.15 per cent before and 0.2 per cent after a meal and with glycosuria, to avoid the diagnosis of diabetes when it actually did not exist.

Sixty-three of the 75 patients seen with diabetes and hyperthyroidism were operated upon with multiple operations being done on many of them (103 operations), with 3 deaths, making an operative mortality of 2.9 per cent and a patient mortality of 4.8 per cent.

There have been no diabetic cures, but there has been an increase in tolerance for carbohydrates not far from 30 gm., and clinically the diabetic course of patients with diabetes and primary hyperthyroidism postoperatively, in Dr. Joslin's opinion, is better than the ordinary diabetic patient with a similar period of hospital treatment.

Hyperthyroidism and Tuberculosis. Little need be said regarding hyperthyroidism and tuberculosis except that removal of the hyperthyroidism so restores the energy and weight balance that the pulmonary infection can be better controlled.

One must realize that the clinical features of tuberculosis in a considerable degree simulate hyperthyroidism, such as myasthenia, tachycardia and weight loss, and that if a coincident nonactive goiter be present these symptoms have not infrequently been wrongly attributed to the goiter, resulting in failure to demonstrate the pulmonary lesion.

The removal of the thyroid in patients with pulmonary tuberculosis should, of course, be done with local anesthesia.

Hyperthyroidism and Cardiac Disease. The association of cardiac disease and hyperthyroidism has been an object of study in our clinic now for a number of years. We have repeatedly reported our experiences with auricular fibrillation and hyperthyroidism. The most recent report may be found in the *American Journal of the Medical Sciences* (1930, clxxix, 507). Dr. L. M. Hurxthal has shown that approximately 10 per cent of all the patients with hyperthy-

roidism show this abnormality. Over 90 per cent of the patients with congestive heart failure and hyperthyroidism have this irregularity, and conversely 30 to 40 per cent of those patients with established auricular fibrillation have, or have shown, various degrees of heart failure.

Dr. Hurxthal advises against the employment of quinidine preoperatively for the restoration of normal rhythm for the following reasons: (1) because patients with thyroidism do not stand quinidine well, and (2) because it will be wasted, since even though the regularity be restored preoperatively, the postoperative reaction to the operation will result in a return of the irregularity and require further treatment in many cases. Quinidine should, therefore, be reserved for restoration of auricular fibrillation to normal rhythm until after the thyroidism has been removed by subtotal thyroidectomy.

He excludes from quinidine therapy any patient who has:

1. A history or clinical evidence of embolism.
2. Marked mitral stenosis with fibrillation of unknown duration.
3. Mitral stenosis with congestive heart failure.
4. Congestive heart failure associated with large feebly pulsating heart as determined by fluoroscopy.
5. Long-standing fibrillation in hyperthyroid patients in whom the hyperthyroidism is of mild degree.

We have always assumed that there was no such a thing as a thyroid heart—that is, a direct destructive effect of the thyroidism upon the heart. We have always felt that cardiac failure and auricular fibrillation were the results of the overdrive forced upon an already damaged heart by the thyroidism rather than that the thyroidism in itself damaged the heart.

The conclusions of Dr. L. M. Hurxthal, who is in charge of the cardiac complications in these patients, will be of interest to you in this connection, and are here appended:

1. If thyrotoxicosis injures the normal healthy heart, we believe there should be more evidence of injury below the age of forty years. The average age of the congestive heart-failure group being above fifty years, with a large percentage showing coëxisting cardiovascular disease, leads us to conclude, on a clinical basis at least, that if there is a selective action of hyperthyroidism it chooses the heart in patients who are over forty years of age. If auricular fibrillation is to be considered evidence of myocardial damage from thyrotoxi-

cosis it certainly should be present more often in the younger patient, and particularly should it have a more definite relationship to duration and degree of toxicity.

2. We believe that thyrotoxicosis has a specific excitatory effect on the heart, and that this, more than the increased work from circulatory demands, produces failure in a heart weakened by the degenerative processes of age.

3. Our clinical observation leads us to believe that hypertrophy of the heart does not take place in a normal healthy heart as the result of hyperthyroidism, but that dilatation is probably frequent. Duration, we feel, plays a very little part in a general way. It is obvious, however, from frequent examinations of thyroid patients, that duration as well as degree of toxicity plays an important rôle in individual cases. Immediate removal of thyroid toxicity by surgical procedure is undoubtedly the quickest and surest method of averting the onset of congestive heart failure.

Therefore, we consider the most significant causes of congestive heart failure in hyperthyroidism:

1. Age and the coëxisting cardiovascular changes associated with it.
2. The specific heart drive incited by thyrotoxicosis.
3. Auricular fibrillation.
4. Duration and intensity of thyroid overactivity.

The diagnosis of thyroidism in the presence of cardiac decompensation is often far from simple. This is due to the fact that the underlying thyroidism which is often the inciting cause of the failure is of the nonactivating type of thyroidism and overshadowed by the urgency of the symptoms associated with decompensation, such as marked orthopnea, ascites, general anasarca and an enlarged and tender liver. Particularly in the absence of exophthalmos and goiter, as is so often the case in the type of thyroidism found in the so-called thyrocardiac, unless particular pains are taken to discover it, an underlying thyroidism can be easily overlooked. One should suspect an underlying thyroidism in any patient with cardiac decompensation when the patient does not regain his or her compensation under the ordinary methods of cardiac treatment, but still does not die as the result of the failure. One should, of course, suspect the presence of an underlying thyroidism in any patient with cardiac decompensation and goiter or exophthalmos. One should suspect an underlying thyroidism in any patient with unexplained

auricular fibrillation, and one should suspect an underlying hyperthyroidism in any patient with a past history of unexplained nervous breakdown with unexplained periods of weight loss.

We have in our writings on this subject stressed the operability of patients with congestive heart failure due to a superimposed hyperthyroidism, and often stated that there are no patients with congestive heart failure of any considerable length of standing due to hyperthyroidism who are too sick to withstand subtotal thyroidectomy with ethylene and with a very reasonable risk in our opinion. The mortality in the so-called thyrocardiac group in our experience has been 3.6 per cent, certainly not excessive in view of the seriousness of the state, and the probable improvement to be anticipated.

We know of no surgical group in which such quick and startling results can be obtained as in the group of patients with cardiac decompensation due to associated hyperthyroidism. The cardiac capacity is often regained within a few days, and patients who previous to the operation were bedridden and unable to recline in bed, not infrequently are able not only to lie flat in bed, but to walk out of the hospital and earn their living within a few weeks of the subtotal thyroidectomy.

In connection with any discussion of cardiac decompensation associated with thyroidism, we have always urged that every patient with intractable heart failure be carefully investigated for the possible presence of an unsuspected underlying thyroidism as the causative factor in failure, the removal of which makes it possible almost to work a miracle in the extent to which cardiac capacity can be restored by the surgical removal of the hyperthyroidism which breaks the compensation.

Hyperthyroidism and Pregnancy. We have been very much interested in the association of hyperthyroidism and pregnancy, and Dr. H. M. Clute, of the clinic, has reported our experiences with this complication in the *American Journal of Medical Sciences* (1930, clxxix, 477).

We have learned in our experience in dealing with such a large number of operated goiter patients that the employment of a general anesthetic and the performance of a general surgical operation in the presence of hyperthyroidism is a very dangerous procedure. We have seen fatalities follow appendectomy, cholecystostomy and perineorraphy in the presence of hyperthyroidism, and have seen hyperthyroidism intensified to a dangerous degree even by tonsillectomy or the removal of a tooth.

It has, therefore, been our attitude that when a patient is seen in pregnancy with hyperthyroidism subtotal thyroidectomy shall be done while patient is still pregnant, rather than the uterus emptied or the operation delayed until after delivery.

The operation of emptying the uterus may seriously intensify the hyperthyroidism, particularly if any infection occurs. If the patient is permitted to go on to full-term delivery, and the labor becomes difficult or requires anesthesia and intervention, serious and fatal hyperthyroidism may result.

Subtotal thyroidectomy can be done in the presence of pregnancy with comparative safety for the mother and child.

In a consecutive group of 3678 patients operated upon for toxic goiter in the clinic, subtotal thyroidectomy was done upon 15 patients with pregnancy and hyperthyroidism, 13 with primary hyperthyroidism (exophthalmic goiter) and 2 with secondary hyperthyroidism (toxic adenomata). There was no mortality, and miscarriage occurred in but 1 case, and in that case two days after a 200-mile automobile ride from the hospital to her home.

We urge, therefore, that in hyperthyroidism associated with pregnancy subtotal thyroidectomy be done during pregnancy and as early in it as possible, and not emptying of the uterus or delay of the thyroidectomy until after delivery.

Since it is the purpose of this lecture primarily to present to you our views on goiter and associated conditions, we shall not enter into a discussion as to the methods of treatment, except to state that we believe that surgery most positively, most completely and most quickly relieves those patients of their thyroidism and with a relatively low degree of risk, the mortality rate for the past few years being here submitted.

TABLE I.—FATALITIES AFTER OPERATIONS FOR HYPERTHYROIDISM IN THE LAHEY CLINIC, 1925–1929 INCLUSIVE

	Primary Hyperthyroidism	
Cases, 2128 Operations, 2739	4 deaths	0.65 per cent 0.51 per cent
	Toxic Adenomatous Goiter	
Cases, 641 Operations, 705	12 deaths	1.87 per cent 1.71 per cent
	All Toxic Cases, Totals	
Cases, 2769 Operations, 3444	26 deaths	0.93 per cent 0.75 per cent

The above mortality rates may be said to be relatively low: (1) when it is appreciated that these figures represent every death in the hospital from any cause whatever; (2) that in the clinic no patients are rejected because of the seriousness of their state; (3) that these figures include even the serious thyrocardiac risks with associated heart failure, and the patients with diabetes and associated hyperthyroidism, each of the latter two groups having a mortality of over 4 per cent.

In mentioning the treatment of hyperthyroidism by surgery, we wish to present our results at the end of a year in terms of basal metabolism estimations, showing the preoperative basal rate, the postoperative basal rate at about eight days after operation and the basal rate at the end of a year.

In Table II is presented the data from 1000 consecutive operated patients with primary hyperthyroidism or exophthalmic goiter, and also the date in 245 consecutive toxic adenoma found in the metabolism records in the consecutive tabulation of the 1000 cases of primary hyperthyroidism.

TABLE II.

	Primary		Secondary	
Lahey Clinic.	No. of cases.	Average.	No. of cases.	Average.
Jewish patients	1000	15%	245	16%
B. M. R. before operation	1000	+49.38	245	+40.94
B. M. R. after operation	1000	+20.48	245	+22.27
B. M. R. 3 months after operation	850	+ 4.85	123	+ 8.62
B. M. R. 6 " " "	652	+ 4.24	81	+ 5.65
B. M. R. 9 " " "	424	+ 2.83	40	+ 5.70
B. M. R. 12 " " "	380	+ 2.34	38	+ 2.63
Gain in weight, from weight after operation:				
3 months after operation	850	15.35 lbs.	123	1.26 lbs.
6 " " "	652	20.43 lbs.	81	7.56 lbs.
9 " " "	424	22.74 lbs.	40	9.43 lbs.
12 " " "	380	24.37 lbs.	38	13.51 lbs.

It will be seen in this table that a progressively diminishing number of patients failed to return for their postoperative basal metabolism check-up until considerably less than one-half of the cases reported for their final basal metabolism estimations at the end of a year.

While the fact that there was a corresponding drop in the basal metabolism rate to normal in both groups of patients, those with primary and those with secondary hyperthyroidism, is noted, the value of the figures is largely negatived by the absence of a report

at the end of a year as to the basal metabolism findings in such a large percentage of the cases. To overcome this weakness in this tabulation, therefore, a series of cases, every one of which had a preoperative and a postoperative basal rate recorded and also one at the end of a year, was sought, and is here submitted in Table III, showing average return to normal rates in 188 patients a year after operation.

TABLE III.—188 CASES OF PRIMARY HYPERTHYROIDISM SHOWING VARIATION IN B. M. R. FROM BEFORE OPERATION TO 12 MONTHS AFTER OPERATION

	Cases.	B. M. R.	Gain in weight, pounds.
Before operation	188	+39	
After operation	188	+15	
Third month after operation	173	+ 7	14
Sixth month after operation	169	+ 2	4
Ninth month after operation	137	+ 1	2
Twelfth month after operation	188	− 3	3

This, we believe, is the method which should be employed in reporting results in any method of treatment in this disease in which basal metabolism rates so typically indicate the presence or absence of thyroid toxicity, uninfluenced by the error of clinical observation and personal equation error.

We urge particularly that any claims for relief of hyperthyroidism by any method of treatment be never submitted in the forms of photographs before and after treatment for two reasons: (1) because a large group of results cannot be submitted this way, and (2) because there is a very definite group of patients with hyperthyroidism who make spontaneous recoveries with or in spite of any form of treatment.

If physicians who are treating hyperthyroidism by nonsurgical measures will report their results at the end of a year, together with their mortality, in unselected and unrejected cases, it will be possible to fairly and honestly compare these methods of treatment each with the other and all with surgery.

I am well aware that there remain several other aspects of goiter and thyroid disease still undiscussed, but the limitations of time and your forbearance, for which I am deeply grateful, prevent their consideration in this lecture.

GALEN*

By CHARLES W. BURR, M.D.

Burton, he of *The Anatomy of Melancholy,* writes: "We can say nothing but what hath been said; the composition and method is ours only. Oribasius, Ætius, Avicenna, have all out of Galen, but to their own method; different styles, but the same reliability." I, likewise, can say nothing new about Galen because he has been written about for eighteeen hundred years by many men of many kinds: geniuses, intellectual humbugs who climbed to fame on the ladder of his knowledge and ignorance; talented men who added to the store of medical knowledge, and simple but honest, learned blockheads. That men of such different types have written about him shows the wide appeal he has made, and thus indirectly proves his importance. Further, I must confess, I have read much more about Galen than in Galen's own works. If, then, you hear echoes of Haeser, Sudhoff, Charles Singer, Garrison, A. J. Brock and a few others, do not severely blame me, but be glad I have chosen such scholars to crib from. My purpose is to show how I, a modern, interested in the history of science, and who believes we can only understand the present by knowing the past, react toward Galen, and to try to draw a picture showing what manner of man he was, and why he was not otherwise. Galen (the name Claudius, he owes to his editors, not to himself or his family), who, all unconsciously, summed up and closed the book of Greek medical science, opened and largely written by Hippocrates, was born in Pergamus in Asia Minor about 130 A.D. He himself said he was thirty-eight years old when Verus died in 169. If he spoke accurately, and not carelessly, this would make 131 his birth year. Some writers claim it was 129. Pergamus was no unimportant city. It was one of the chief centers of the worship of Asclepius, possessed a great temple dedicated to Zeus, was an early focus of Christianity, the seat of one of "the Seven Churches," and, most wonderful of all, it was, accord-

* Read at the Galen Memorial Meeting, Philadelphia College of Physicians, October 23, 1930.

ing to the *Book of Revelation*, the place of "Satan's Seat." Zeus, in St. John's belief (I believe he is still accepted as the author), was Satan, but whether some theologic quarrel, or damned heresy, also influenced him, in giving such black immortality to the city, I do not know. But it survives, and shows today no more signs of Satanic influence than most towns, and less than many. Perhaps Satan has moved a few thousand miles west.

Galen's father, Nicon, was a man of substance, virtuous in the Roman, manly sense, learned in philosophy, astronomy and geometry, and skilled in his profession, architecture. His mother, though virtuous, had an acid tongue and a bad temper (she punished her maids by biting them and sometimes bit when no punishment was deserved) and took Xanthippe for her model, especially in her treatment of her husband. Temper and tongue were inherited by the son, but in less degree. He was a quarrelsome man, as is shown in his writings, had no sign of an inferiority complex and was industrious and ambitious. His intellectual talents he inherited entirely, so far as we know, from his father.

We know nothing of Galen's ancestry, and only the little about his parents I have stated. The ancients seem to have had little interest in the study of heredity, or rather, regarded it as so self evident as not to require proof. They were more interested in knowing what men did or thought than in the why or how. Psychology, as we think of it, the study of mind as revealed by behavior, and its causes, hereditary and environmental, was unborn until almost the other day. Galen had biologically good parents, though today some psychologists would claim endocrine dysbalance in his mother, and had there been Freudians in those days, they would have loved to have psychoanalyzed her and have attended to her *libido*. The mental environment of his childhood and youth, the atmosphere in which he lived, save his mother's vicious temper, was excellent; the material was such as to make possible every purchasable advantage. He had good teachers, as a boy and young man, and was well grounded in Aristotelian and other philosophy. He studied under Stoics, Academics, Peripatetics and Epicureans. Apparently, he was led, when about seventeen years of age, to study medicine by a dream his father had. His interpretation of it was not by Freudian methods, but, accepting the correctness of his premises as to what dreams are, was more useful than Freudian interpretations usually are, and, of course, was based on a cleaner

psychology. He traveled much while a student, indeed through his life, and had eminent teachers in Smyrna, Corinth and at last Alexandria.

I am going to recall to you an incident of Galen's time, though he was not in Smyrna when it happened, because it illustrates the ferment which had already begun to work in the minds of people, and the unrest, both civil and religious, present throughout the empire. I refer to the martyrdom of Polycarp. A great public festival was being held in 155. Christian persecution was widespread. Bishop Polycarp, who was a connecting link with apostolic times and a great upholder of the true faith, undiluted by those terrible things, heresy and false doctrine, was brought before the proconsul as a result of the tumultous rioting arising from the celebration, and was asked by him to abjure his faith to save his life. He replied: "Eighty and six years have I served Him and He hath done me no harm. How then can I speak evil of my king, who saved me?" The mob demanded the life of Polycarp and got its wish, but whether Galen heard of the incident we do not know. He would have paid little attention to it, just as, I suspect, we would under similar conditions; but it illustrates the nature of the time in which he lived, and that influences every man, not only consciously, but unconsciously.

Alexandria, where he ended his student days, was a great center of learning and trade: it fed the bodies of the Romans with grain, and the minds of all men with science and learning. All races met there, and philosophers of all schools studied, thought, taught, discussed, dreamed and quarreled. He must many times have seen the monument called Cleopatra's needle, now in New York, and its companion, now in London. He may have meditated, sitting in their shadow. In Alexandria he had opportunity to study the bones of the human skeleton. He also learned Latin, Persian and several other languages. He remained there several years and returned to Pergamus to practice when twenty-eight years of age. Soon he was officially put in medical charge of wounded gladiators. He did well, especially in cases of nerve injuries; but, sometime later, there were serious riots in the town, and he, never a courageous man, fled to Rome. Arrived there, he largely gave up surgery and devoted himself to medicine. He was successful and, as frequently happens, success brought with it enmity, envy and hate. His enemies called him theorist, a surgeon with his tongue rather than with his hands

and head, and even claimed he was a sorcerer, making some of his cures—and, apparently, he made great cures—by black magic. He was not in sympathy with magic and magicians. Astrology, also, which was a fundamental belief of the older Stoics, made no appeal to him, and he says that in his time, in Rome, the people only bothered about it in forecasting legacies. I suspect the populace sought astrological advice as to lucky days. The populace remain the same today. Even if Galen had not been a conceited and quarrelsome man, he would not have had a peaceful time in Rome, because medicine was on its way toward intellectual chaos there, and almost every physician fought bitterly the members of rival schools. There were many schools—Methodists, Dogmatists, Pneumatists, Eclectics and others—all calling each other names, and few of the members of any school were really skilled. Open quackery flourished even more than today. Given, then, a man learned in philosophy, learned and skilled in medicine, anxious to continue learning, a talented mind, the reverse of modest, ambitious and, above all, successful, quarreling was inevitable. He joined no school and was a real Eclectic, taking knowledge wherever he found it. The quarrels, coupled with a plague which was ravaging Italy, led to his leaving Rome again and returning to Pergamus, which had once more become quiet. Later the Emperors Marcus Aurelius and Lucius Verus, knowing his great talent, sent for him. He, obedient to imperial orders, went. They meanwhile left Aquilia, where they had been on account of a plague, and Verus died on the journey and Galen returned to Rome. Soon after Marcus Aurelius requested him to go with him, as a physician, on a military expedition against some German tribes. He did not wish to go and, opportunely, had a dream in which Æsculapius appeared and told him to remain in Rome. He related the dream to the Emperor, who accepted it as a sufficient reason and ordered Galen to remain in Rome and to take medical care of his son, Commodus. During this period an unfortunate accident happened: Galen gave lectures on anatomy at the temple dedicated to peace, and also had deposited there many manuscripts of his writings. The temple caught fire and the papers were burnt. His reputation continued to grow, but also the hate and envy of many doctors, and he left Rome. Of his later years we know little. There has been much dispute as to where and when he died, but he certainly was dead by 200 A.D.

So much for the externals, the events, in Galen's life. Now let us take up his work and try to discover what kind of man he was. He had a Greek mind, was credulous, but not, considering his age, superstitious. The man whose medical writings influenced Galen most, Hippocrates, cared little for theory, for philosophy, was practical minded and not philosophical. Galen had from youth on studied Aristotle and was greatly influenced by his writings. Hippocrates and Aristotle were Galen's two intellectual masters: they did not nullify, they supplemented each other. He had a hunger, which Aristole fed, for seeking the reason of things. Also, Aristotle's belief that there was a God back of all things, and that the universe was designed and not merely the result of the accidental clashing of atoms in space, as the Stoics believed, appealed greatly to Galen. Nature, to him, was an artist, with God back of and controlling it. Had he reappeared in the world in the early nineteenth century, the Bridgewater treatises, written to prove design, would not only have appealed to him, but he would have understood the point of view just as quickly and just as sympathetically as he would, for other reasons, have appreciated the work of Harvey on the circulation of the blood.

Let me recall, I trust it will not bore you, the theories underlying Galenic medicine. I borrow, for this purpose, from that mine of now useless and forgotten but ever-interesting medical lore, Dr. James' *Medical Dictionary,* published in three huge folio volumes in 1743 to 1745. Even as late as that date, Galen is referred to and quoted more frequently than any other man. James, and the physicians of his time, of course, realized many of his errors, but still his spirit had profound influence. Galen accepted from Hippocrates the four humors—the blood, the phlegm, yellow bile and black bile—and the idea of hotness, coldness, wetness and dryness of these humors; *e. g.*, blood was red, hot and moist; phlegm white, cold and moist.

The body was made of parts, humors, spirits (pneuma). There were three kinds of spirits: (1) The natural, (2) the vital, (3) the animal. These three served as instruments of three faculties. (I need not remind you, by spirits, he does not mean bodiless entities, but an essence pervading things.)

1. The natural spirit was subtle fluid arising from the blood in the liver. It presided over nutrition, growth and generation of the animal. It ebbed and flowed through the veins.

2. The vital arose in the heart and, passing as a fluid through the arteries, it gave warmth and life to all the body.

3. The animal, the noblest, had its seat in the brain; reasoning depended on it and by means of the nerves the spirit gave motion and sensation and presided over all other faculties. (The animal spirits flow back and forth through channels in the nerves, which become obliterated in death.)

Now these three faculties produced three sorts of actions: the natural, the vital, the animal. These actions were again divided into external and internal.

1. Internal actions of the animal. Imagination, reasoning, memory.

2. External. The five senses, general sensation and motion.

Internal actions of the vital faculty are violent passions, *e. g.*, anger. External action, the pulsation of the arteries and movement of blood.

The internal actions of the natural faculty are bloodmaking, concoction of food and concupiscence. The external actions are the distribution of blood to all parts by the veins, thus serving nutrition, growth and preservation of the body and propagation of the species. A man was in health when the faculties performed their ordinary actions. Actions cannot be performed healthily unless parts and humors are healthy. If parts or humors are not acting well, the spirits run into disorderly motions and disease results.

Causation of disease: (1) External; (2) internal.

External. Six.—air, food and drink, motion and rest, sleeping and watching, retention and excretion, and the passions.

The external causes put in motion; the internal, of which there are two kinds: (1) The antecedent; (2) the conjunct.

1. Depends upon a peccancy of the humors, with a plethora, or a bad state of one or more of them.

2. Is more closely connected with the disease itself and if removed cures it, *e. g.*, in pleurisy, the conjunct cause is the humor adhering to the pleura and causing the inflammation.

Though Galen knew that he knew nothing of the causes of many diseases, yet his therapeutics was based largely on his theories. But his greatness, as a mind, depends on other things altogether than his theorizing; it is shown by his work in experimental physiology and his anatomical studies, though his experiments were neglected for centuries. An excellent example of his work in phy-

siology is his experiment to prove that urine enters the bladder by the ureters. I quote the account of it from Brock's translation. (Galen, *On the Natural Faculties.*)

"One has to divide the peritoneum in front of the ureters, then secure these with ligatures, and next, having bandaged up the animal, let him go (for he will not continue to urinate). After this, one loosens the external bandage and shows the bladder empty and the ureters quite full and distended—in fact, almost on the point of rupturing; on removing the ligature from them, one then plainly sees the bladder becoming filled with urine.

"When this has been made quite clear, then, before the animal urinates, one has to tie a ligature round his penis and then to squeeze the bladder all over; still nothing goes back through the ureters to the kidneys. Here, then, it becomes obvious that not only in a dead animal, but in one which is still living, the ureters are prevented from receiving back the urine from the bladder. These observations having been made, one now loosens the ligature from the animal's penis and allows him to urinate, then again ligatures one of the ureters and leaves the other to discharge into the bladder. Allowing, then, some time to elapse, one now demonstrates that the ureter which was ligatured is obviously full and distended on the side next to the kidneys, while the other one—that from which the ligature had been taken—is itself flaccid, but has filled the bladder with urine. Then, again, one must divide the full ureter, and demonstrate how the urine spurts out of it, like blood in the operation of venesection; and after this one cuts through the other also, and both being thus divided, one bandages up the animal externally. Then when enough time seems to have elapsed, one takes off the bandages; the bladder will now be found empty, and the whole region between the intestines and the peritoneum full of urine, as if the animal were suffering from dropsy."

As said, his study of anatomy was made on Barbary apes and other lower animals, with occasional opportunity to examine human skeletons or stray bones. Dissection of the human body was forbidden throughout the empire. He had opportunity to study the human skeleton in Alexandria. His account of the skeleton and its component bones is, therefore, the best of his purely anatomic work, though he regarded the teeth as bones. He never had opportunity (as far as I know) of studying human musculature in general. Using Barbary apes, he distinguished between the origins and insertions of muscles, and knew the function of many. His nervous anatomy was excellent. From the study of the brains of oxen, he distinguished the dura and pia mater, the corpus callosum, the third and fourth ventricles, with the aqueduct of Sylvius, the fornix, corpora quadrigemina, vermiform process, calamus scriptorius,

hypophysis and infundibulum. He knew seven pairs of the twelve cerebral nerves, and described the sympathetic ganglia, which he regarded as the reinforcers of the nerves. He was the first experimental neurologist. He proved that all nerves arise from the brain or spinal cord, and separated the sensory from the motor functions. He was the first to make experimental sections of the spinal cord, and knew the different results depending on difference in level. He caused aphonia by cutting the recurrent laryngeal nerve. He learned, by watching the heart of animals excised at sacrificial ceremonies, that it continued to beat when removed from the body and realized that this meant, its pulsation was not dependent upon outside mechanism. He made one observation on voluntary muscles, which gave him a very modern concept, that of muscle tonus as causing active posture. He had some notion of the reciprocal action of antagonistic muscles. He made some curious anatomical mistakes, many, if not all, of which were due to the fact that he studied not man's anatomy but that of animals; *e. g.*, the uterus, according to him, is two-chambered, and he explained the sex of children by making the right testicle and right cavity of the womb the cause of male offspring.

I shall say nothing of his work on the eyes, and the theory of vision he accepted. Dr. Chance will tell you about it.

He, of course, knew nothing of the circulation of the blood, but had he been able to clear his mind of prejudgment, he might have made the discovery. He drew the conclusion that there was anastomosis between arteries and veins, because, as he writes: "If you kill an animal by cutting through a number of its large arteries, you will find the veins becoming empty along with the arteries; now, this could never occur if there was not anastomosis between them." Harvey never saw the link between arteries and veins, but wisely guessed it was there. Galen did not appreciate the importance of his own conclusion. Again, Galen looked closely for the perforations in the cardiac septa (that the medical philosophy of his day claimed were there, thus explaining how blood got from one side of the heart to the other), but could not find them. He did not trust his own eyes as against dogma, but upheld dogma, explaining the pores were there, but too small to see.

He recognized phthisis as a disease, and knew the therapeutic importance of climate and feeding, especially milk. Parenthetically, let me remind you, the Romans paid much attention to hygiene,

both public and private. He differentiated pneumonia from pleurisy, and was the first to describe aneurysm. He described what must have been encephalitis lethargica.

He was always (like his master in philosophy, Aristotle) hunting for the why of things, never content with learning the how. He was very modern in some things. Thus, he discusses the question as to whether structure follows or precedes function, a matter still discussed, and, I think, the following quotation shows his opinion is that of many biologists of today, though his proofs belong largely to a now-dead world. He writes: "Thus man is the most intelligent of all animals; thus the hands are proper instruments for an intelligent being, since man is not wiser than the animals because he has hands (as Anaxagoras maintains), but as the judicious Aristotle asserts, he has hands precisely because he is the wisest. It is not by means of his hands, in fact, but through his reason that man learned the arts; the hands are an instrument like the lyre to the musician, like the tongs to a blacksmith. . . If we examine newly born creatures that strive to act before the parts of the body are fully formed, it becomes clear that it is not the parts of the body which excite the soul to be cowardly, courageous or wise. For example, I have often seen a calf try to gore before its horns had developed, a chicken try to spur although its feet were soft, and a little pig trying to bite without teeth; for every animal has in himself, without any previous instruction, an instinctive feeling of the faculties and functions of his body. . . It is not through instruction, I opine, that the eagle soars, the duck swims and the snake glides into a hole, for as Hippocrates says, animal natures are untaught. Whence it seems to me, for the rest, that animals practise certain arts more by instinct than through reason."

What considered judgment can we pass on Galen? One thing makes a man, his heredity; another molds him, if he is moldable, his environment; the two combined give the final result. Galen's heredity was, all that we know of it, good; even his mother's bad temper may not have been an unmixed evil. The environment, so far as his parents willed it, was wise, and so far as he willed it, he accepted and chose all the good that came to him, in teachers and travel and work and study. The environment that could not be controlled—the time of living, the second century; the place, the Roman Empire—was not the best, but he seized all it offered. The second century A.D. was not an ideal time to develop men of

science or men of art. Superficially looked at, the Roman Empire was still great, but already the seeds of decay had been planted. Roman citizenship was not made up of the little caste of former times, men of one race, one background, one tradition, but included men of different races and several levels of culture. In religion, the old gods were dead or dying; the new religion was struggling through a sad and perilous birth, and many of its midwives were far from wise, and some not virtuous. Economically, the growth of the empire brought great wealth to many who did not know how to use it, having no background, and this led to much evil. Rome was never the breeding ground of art and science, as Greece had been. Creature comfort was destroying the old Roman idea of virtue, and grafters and professional reformers were destroying statecraft. Malaria had already begun to undermine the people's health. Into this atmosphere Galen was born. Education and inherent tendency made him hold firmly to Aristotle and his God who designed and rules the Universe. This colored, indeed controlled, his point of view toward life, science, philosophy. Accident seems to have made him a physician. Intellectual curiosity drew him to the study of the works of the great master of observation, Hippocrates, whose memory he revered even when he disagreed with, even contradicted, his opinion. But, and it seems to me his only real mental fault, he got from someone in his ancestry, the craving for theorizing and hyposthetizing. This craving may have been encouraged by some of his teachers of philosophy, not Aristotle, who was a great observer. He had to know, or think he knew, the why of things. Hence, sometimes, instead of perceiving what he looked at, he saw what he wished to see.

He, undoubtedly, was the great physician of his period, though some of his contemporaries did not think so. Worldwide fame came later and grew with time. The Emperor Julian (331–363), a great and honest man, who has been damned by all priestcraft since his time, because, when he reached maturity, he discarded the doctrines forced on him as a child and immature youth, helped him to immortality by having Oribasius edit his many works. It goes without saying that the basis of Galen's fame and influence was his medical work, especially his system of therapeutics; but there was another factor, the church, as Singer points out. The church, especially in medieval times, helped to increase his fame, partly because the philosophy of Aristotle, which so greatly influenced him, was

largely accepted and adapted by it. The Arabs, who added little to science in their short period, and the scholarly Jews, accepted his teaching and preserved his works in manuscript, when the Empire was destroyed and Europe so in chaos, so saturated with the idea that this life was but a way station between two eternities, so overwhelmed with the saving of individual souls that all science ceased, save here and there in stray corners, where single scholars surreptitiously carried on. For centuries the human body was regarded as too contemptible to be worth study, but also too divine to dissect. Professors of anatomy were too dignified to dirty their fingers. I do not wish to imply that the growth in power of church philosophy was the one cause of the cessation of growth of scientific knowledge. It was only one of many causes, but a potent cause.

Then came the Renaissance, thirteen centuries after Galen's death, and Leonardo da Vinci (Vesalius' forerunner), who probably never read Galen, but must have heard much of him, because all medical learning was still saturated with him, studied anatomy itself, and shortly after Vesalius made the great actual study of the human body. Paracelsus burnt Galen's books, as a symbol of getting back to nature. Humbug and charlatan as he was, yet a thinker and doer, I sometimes wonder if this act was not an honest one, and if he did not really mean, study bodies and not books, sick men, and not what writers say about disease. The dethronement of Galen began with Vesalius, because then his infallibility began to be doubted, but was not completed until Listerism won. Thus Galen's teaching, that suppuration was an essential part of the healing of wounds, and pus laudable, continued to be accepted through the ages, though a few preached against it, even in early times—Paracelsus, Paré and a few others—until Lister showed the medical world that all pus was evil. That was the end of Galenism, though even as recently as my student days, the word alterative was still used in the books on therapeutics as the name of one class of remedies, and an alterative is, in the old dictionaries, defined as a drug which alters the humors. But Galen still remains, and ever will, as the man who summarized all Greek medical thought, adding thereto a worthwhile portion of real discovery. It was not his fault that the world for centuries accepted his hypotheses and his therapeutics as final, but neglected to learn and to use the greatest lesson he taught, the importance of experimental research.

GALEN'S OPHTHALMOLOGY*

By BURTON CHANCE, M.D.

Dr. Welch and Dr. Burr have told us of the greatness of Galen in the world of medicine and the wide fields of learning and philosophy. I, too, desire to render homage to that Prince, whose dicta tyrannously dominated the "Province of Ophthalmology" for 1500 years.

When, at about the middle of the second century, the huge, overshadowing figure of Galen arose upon the medical world, the ancient period of civilization was ending and its glories fading from the minds of practitioners. In the Roman world upon which he looked, science had almost ceased to exist; the profession debased; commercialism and quackery ruled, and decency was almost unknown. In spite of all this, Galen went on with his working and his writings, and in the enunciation of his inordinate theorizing, which in time was destined to so profoundly affect the world that his influence extended and endured here and there until almost the beginning of the last century. Under his influence Graeco-Roman ophthalmology far exceeded that of any predecessor and was continuous until the discovery of the true function of the crystalline lens.

Let us for a few moments speak of the main discoveries of the earlier times. Throughout the remote ages of antiquity the eye as the organ of vision was regarded with awe and shrouded in mystery. By the time of Hammurabi, of Babylon (2250 B.C.), a distinct money value was placed upon that organ, and laws regulating the practice of surgeons undertaking the care of the sick were imposed and penalties proscribed for failure to cure the diseased eye. Nevertheless, in general the eye was treated with the crudest superstition and subjected to ridiculous magic.

In somewhat later years the practitioners of Egypt had advanced greatly and enjoyed a high reputation. Items relating to ophthalmic surgery are mentioned in the early texts, as in the Ebers' Papyrus, which dates from the Seventeenth Century B.C., yet

* Read at the Galen Memorial Meeting, Philadelphia College of Physicians, October 23, 1930.

outside these definitions the customs of that country were equally absurd and magical. Some twelve centuries later, the Alexandrians added something to the knowledge concerning the eye and its appendages; they were assiduous dissectors and had studied the body of man as well as those of various animals. The "Father of Scientific Anatomy," Herophilus, in the Fourth Century B.C., proved himself a precise dissector, for he was able to show the relations of the larger nerves to the brain and the spinal cord, and it was he who distinguished the motor nerves from the sensory. He was the first to describe the lens, the ciliary body, the retina and the vitreous. For two or three centuries longer the scientific labors of the Alexandrians continued at a high level.

In Greece the greatest advance is noted in the Hippocratic writings, dating from the Fifth Century B.C., which largely record the traditions and teachings of centuries long anterior to Hippocrates. In the attempts to describe the structure of the eye, it is noted that the eye consists of three membranes, the white, the soft and the spiderweb-like; and these membranes are distended by the three ocular humors; the aqueous, the vitreous and the crystalline. The optic nerve was known, although the function of it was not understood. In that age it was believed that the essential organ of sight was not the retina and nerve, but either the vitreous or the crystalline.

In the treatment of eye diseases, Hippocrates prescribed restriction of the diet, hot foot-baths, cupping and venesection. As a favorite remedy he would order the burning out of the bloodvessels in the temple or forehead; and, to afford an exit to the morbid "humors" of the eye, deep incisions were made in various portions of the scalp. Throughout the Mediterranean countries, even at that remote time, trachoma existed and it was the most prolific cause of eye affections. The granulations were treated by a process called "blepharoxysis," which consisted of rubbing the inner surfaces of the eyelids with wool wrapped about a stick of wood.

Roman medicine was almost entirely carried on by Greeks, as it was considered demeaning for a citizen to practise medicine. At the end of the First Century of our era medical practitioners were guided by the works of Celsus, certain chapters in his Sixth Book being devoted to the eye and its diseases. These works were more or less handbooks for the laity, being adaptations from Hippocrates and other sources, yet in many respects they show an advance

beyond the Hippocratic position. Dietetic regulations, venesections, hot poultices made from bread and honey and other articles from the domestic dispensatory or pharmacopeia were prescribed, while crude surgical incisions and dissections with the free use of cauteries were practised.

The exact position and nature of cataract in the time of Celsus were unknown. The efforts at removal of such obstructions to sight were indeed limited and were commonly unavailing, as they consisted simply of couching. From the days of Hippocrates it was supposed that a wide space existed between the iris and the lens, and that in cataract formation this space became filled with a thickened humor which flowed down into it from above. The "coucher" of those days believed that the needle caught this humor and depressed it out of the line of vision, while the lens itself remained undisturbed. These ideas and practices were held continuously until the Eighteenth Century, and are still prevalent in certain parts of India.

Galen was taught philosophy and medicine first in the school of Pergamus. In his travels about the lands bordering on the Mediterranean he had observed and noted the practice of the physicians of the great schools of Smyrna, Corinth and Alexandria, and his works are epitomes of what he had learnt and seen, representing a treasure house of medical knowledge and experience, custom, tradition and history. They, like the Hippocratic corpus, were made up of materials gathered from various sources, in the makeup of which Galen acknowledged his indebtedness to the Hippocratic writings.

Out of the enormous mass of the works of this man, a detailed list of which has been preserved, certain have been rescued from destruction. Whether these be truly Galen's own or are spurious, it is still difficult to say; suffice it, however, that they have been accepted through the ages. The strictly ophthalmological writings, *Optics*, and *Diagnostics of Diseases of the Eye*, to which reference is repeatedly made, have been lost. It is not unlikely that some Arabian translator failed to publish his product as a work of Galen's, and it was not impossible for one of the famous Easterns to pass off the work as his own during the centuries that were dominated by the Arabians, so difficult is it to trace ancient works through all the courses of translation and retranslation.

The two works of interest to us are *On the Use of the Bodily Parts*

in Man and of the *Treatment of Ophthalmic Affections*. The organ of vision is considered, its parts described and an hypothesis given on the subject of vision. The work on therapeutics, which is regarded as spurious, also gives an account of the eyes, their construction, their tunics, humors, muscles, nerves, etc., the mode in which vision is accomplished. The affections of the eyes and of their respective parts are considered, and the remedial measures to be adopted for their relief. An immense assortment of "Collyria" is presented.

The eyes were looked upon as directly connected with the brain by hard and tough processes, which expand and surround the vitreous humor on all sides and become attached to the crystalline body. The crystalline body was credited with being the essential organ of vision, as was proved when hypochymata, or cataract, formed between the crystalline humor and the cornea, which thereby took away the sight. The vitreous body, which resembles molten glass, nourishes the lens, while the vitreous itself is nourished by the structure which surrounds it and which is sent down from the brain to protect it. This is the "net-like" membrane. A vague idea of nerve connection and conduction was discerned, for it was believed that this membrane perceived the alterations which occurred in the crystalline and informed the brain concerning them. This retina is surrounded by the choroid coat, full of arteries and veins, which also takes its origin in the brain and which in its turn nourishes the retina. Covering the sclera is a membrance which acts like a tendonous sheet from the muscles which move the eye.

"There comes to the eye an outgrowth from the periosteum of the orbit which not only unites the eye with the bones, but serves to protect the muscles." This, of course, implies the conjunctiva; yet we now know the conjunctiva is not derived from the orbital periosteum. "It is a soft structure which ends where all the circles lie beneath, where, in fact, the 'black and the white' come together." This place is called the "iris;" it was to Galen a "place," and not the membraneous optical diaphragm we know as the iris, with its pupil. Galen's name for the structure was "hragoeides," a grape skin.

"There must be a protective and also a transparent cover or front for the eye; this also takes its origin from the hard sheet from the brain. It is a small, thick disk, cut, as it were, out of clear horn, which those learned in anatomy called the 'cornea.' A thin fluid

lies between the crystal and the grapelike membrane. The pupil or hole in the grapelike skin is filled with air."

As to his ocular physiology, which is based on his anatomy, rays (called "visual rays") proceed from the brain, down the optic nerve to the retina, from the retina to the crystalline body, and then from the crystalline body out through the pupil, through the aqueous, cornea and so on, in exactly straight lines to the object looked at. The rays then return by precisely the same path which they took in the outward direction, and, reëntering through cornea, pneuma and aqueous, reach the crystalline body or essential organ of vision. Here vision is in some mysterious manner manufactured or elaborated and then transmitted, by means of the retina and nerve, to the brain.

Galen acquired much from the Alexandrians, who were famous for the accuracy of their observations, and he borrowed with a free hand. To Herophilus he was deeply indebted. In judging of Galen's "Anatomy" one must bear in mind continually the little that was written by Hippocrates, the less that was left by Aristotle, and even the somewhat slighter amount set down by Rufus of Ephesus, of whom Galen speaks with respect and as though he were a recent author. Rufus, who was the author of the first formal work on human anatomy, details in his description of the eye his discovery of the optic chiasm.

The ocular anatomy and physiology of Galen are the very highest development, indeed, the culmination of knowledge of these two subjects in all antiquity, and nothing of any great worth was added to them for more than 1500 years, indeed, not until the time of Brisseau and Maitre Jan.

It is apparent to us of today, that in not a few places his anatomy was only that of animals. As the dead body was held most sacred and not to be defiled, dissection of man was then out of the question among the Romans. Galen, therefore, was forced to dissect apes and pigs, dogs and other animals. From his dissection of the brains of oxen he distinguished the dural and pial membranes and a number of the divisions and sections of the brain. He was the first to describe the cranial nerves. His mistakes were dominant for 1400 years. Thus the crystalline lens is placed in the middle of the eyeball and is made the principal organ of vision. The other tunics and humors are supposed to protect and nourish the lens. The retina is recognized as the terminal extension of the optic nerve,

but its true function is unknown. He could not know that each optic nerve contained fibers from the two sides of the brain; the direct, and those which come from the opposite side crossing in the chiasm, which is one of the great bands connecting the two halves of the cerebrum. The six muscles of the eye are well described, but to them is added a retractor bulbi, a muscle which is found only in certain animals. All were repeated by the great anatomist, Vesalius, in the middle of the Sixteenth Century! It was Falloppio who proved that the retractor bulbi did not exist in the human eye; and, in 1600, Fabricius defined the true position of the lens. In 1604 Kepler discovered the true nature of the lens and retina. Galen subordinated accurate descriptions of structures to speculations about the functions. He was the first and foremost contributor to experimental physiology before Harvey and the first experimental neurologist.

The various sections of these works discuss the respective signs and symptoms of the disorders of which they treat, but necessarily with no morbid anatomy. Galen was the greatest of theorists and his pathology was founded upon the theory of temperaments, and thus upon the Hippocratic doctrine of humors and the Pythagorean theory of the four elements, what were afterward denominated as the "sanguine, choleric, melancholic and phlegmatic." He had been nurtured in the Hippocratic tradition, to which he was devotedly attached.

Galen's ocular pathology did not differ much from that of Celsus. In his remarks on "Cataract," there occurs this striking passage:

> "Among the oculists of our day, a certain Justus has cured numerous patients of their hypopyon by shaking their heads. He places the patient upright on a chair, grasps the head on opposite sides, and shakes it until the pus runs down before our eyes. It then remains below, because of the heaviness of its substance. On the other hand, a cataract does not remain below (after it is couched) unless one carefully fastens it down."

This indicates that Galen did not understand the essential difference between hypochyma (or cararact) and hypopyon.

Galen was the most skilled practitioner of his time, yet he left no good accounts of his clinical cases, but only his miraculous cures, for he usually got his patients well. He emphasized, as did all the Greek schools, the duty of treating the individual, and not narrowly, only the local lesions or disease. In his therapeutics he was decidedly in advance of any of the earlier writers.

His principle was cure by contraries, and his drugs were classified by the four qualities, hot, cold, moist and dry. He practised a most elaborate system of polypharmacy, avoiding minerals, especially mercury, relying upon plants and vegetables, the lists of which formed the Galenical herbals. He traveled far to learn all he could about the native remedies of the different regions.

From his knowledge of anatomy he must have been a good surgeon; he insisted upon the clearing of all pockets about a wound and on the securing of clean and free drainage therefrom. It was the custom in his time, however, to send surgical cases to others, known as professional surgeons; a custom to which Galen had to submit. Surgical instrumentation was highly specialized and plastic surgery as well as operations for cataract were skilfully performed. And, under the Romans, ophthalmic surgery attained a degree of perfection which it was not to reach again before the time of Ambroise Paré.

It would be inappropriate for me on this occasion to detail the descriptions of each of the parts and each disease of the eye recorded by Galen; only a general sketch is permissible. Much, therefore, has been omitted by me that would otherwise be of absorbing interest to the Section of this College devoted to the study of the eye and its affections. It is only that I would draw your attention to the efforts made, nearly 2000 years ago, by one who sought to learn of the structure of man and to relieve his distresses. I need not tell you that what I have read to you tonight has been gathered from many sources from the interesting books in the College Library.

The ophthalmology of Galen very largely prevailed in the civilized world from the time of his death on through the Middle Ages and into the modern period, almost as far as the middle of the Eighteenth Century, and he continued to be, just as in every other department of European medicine, the dictator for nearly fourteen centuries until the revival of learning and beyond. As Meyer aptly says, "for Rome, Galen was an Episode; for the Middle Ages, an Epoch."

THE ORIGIN OF THE UNIVERSITY OF BOLOGNA (ITALY)*

By GIUSEPPE FRANCHINI, M.D.
PROFESSOR OF TROPICAL DISEASES, UNIVERSITY OF BOLOGNA, ITALY.

The fall of the Roman Empire, which had reached a very high degree of civilization, determined the complex phenomenon of civil decadence that found favorable development in Christianity's teachings of the renunciation.

The dark ages followed, during which hordes of hungry and bloodthirsty barbarians took advantage and invaded Italy, being attracted by its temperate climate and the opulence of the fields. Goths, Huns, Vandals and Longbards descended from the northern steppes and made of Italy a battlefield of passions and plunders; they were real barbarians, not in the strict Latin meaning of the word, but because of their lack of every form of civilization. This *flagellum Dei* (scourge of God) was in open contrast to the Latin domination over Hellas and the Hellenic occupation of the Pharaonic Empire, where the conquerors were won by the civilization of the subdued people. In their case literature and the arts flourished among them, and found, through the new rulers, not only greatness but further impulse toward progress.

While the Ptolemys, succeeding Alexander the Great, were creating a cultural center in Alexandria of Egypt, the northern barbarians were scourging the peninsula with constant conflagrations and plunders. Whatever part of the immense Greek-Roman intellectual patrimony we now possess we owe to those who, still burning with the sacred fire of the imperial civilization, met secretly in churches and monasteries or in secluded woods and kept their culture alive. It is, however, well known that Theodoric, king of the Ostrogoths, though he himself could not read or write, protected Cassiodorus, Ennodius and Boethius, who cultivated the Roman literature, while he himself took pride in erecting magnificent edifices.

* Read at the Galen Memorial Meeting, Philadelphia College of Physicians, October 23, 1930.

At Bologna, the "School," so-called for lack of better term, was established at the initiative of a few cultured men; it was entirely private, without any control of the princes who, given to warfare, despised any and every form of activity foreign to military affairs. Literature was not considered an occupation fit for a man of the world and, consequently, it could be pursued only by the clergy. Since everything centered in theology, scholars shut themselves in the inviolable recesses of religious communities where peace and security turned their mind to meditation. Abelard's and Friar Benedict's schools at Troyes originated in this way. The Cenacle of Montecassino in the fifth century, the first example in Italy, perhaps, of organized study with magisterial directions, closely simulated the Justinian school of law. Saint Benedict's cenobites did not disdain to teach the medical art, being possibly the forerunners of famous teachers of the Salernitan School. In the year 802, Charles the Great, in the first Imperial Edict, made of Salerno, already a well-renowned school of medical discipline, the best-known center of education. This edict was the only one of its kind until four centuries later. Dante reminds us that Parisian schools were confined to *vicus straminis* (Straw Street), where teachers and pupils sat on straw in curious places in which *in parte superiore magistri legebant, in inferiore meretrices officia turpitudinis exercebant* and *inter se et cum hominibus litigabant* (on the second floor teachers lectured and on the first prostitutes practised their obnoxious business . . . and wrangled with men and among themselves.

The Studio of Bologna. Bologna did not enjoy any greater privileges. Here, as elsewhere, teaching began as a private initiative, and Odophredus cites *quidam dominus Pepo* (a certain Sir Pepo) who *cepit auctoritate sua legere in legibus* (began with his authority to read in the laws), and was soon followed by Yrnerius with the same teaching. In the year 1000, as H. Rashdall reminds us, "the professor was not originally the officer of any public institution: he was simply a private-adventure lecturer—like the sophist of ancient Greece or the rhetor of ancient Rome—whom a number of independent gentlemen of all ages between seventeen and forty had hired to instruct them."

The School of Bologna, thus formed, developed and flourished as a private institution, not, however, lodged in ill-famed houses, as in Paris. In the fifteenth century we find artists studying in different places from lawyers, the use of straw, far from being banned

must have been pragmatic, since as late as 1405 it served the students as an argument in favor of anticipating Christmas vacations, which were demanded *proiciendo paleas vel faciendo aliquem actum per quem impediatur doctorem legere* (by throwing straws or by doing some other act to prevent the doctor from lecturing). To this very year (1405) belongs the first statute of the College of Medicine and of Arts, that prohibited lecturing in other localities than those expressly indicated and listed, in the vicinity of which no noisy business or profession could be carried on. Students paid the teacher a fee *pro suo salario et collecta bancharum* (for his salary for collecting funds) and were brought together in hospices or colleges where, through statutory provisions going back to the year 1284, they enjoyed every sort of privilege and protection. The hospitality offered students and their respective families by the city governments was generous indeed, and it aimed at making possible for the University a more vigorous growth *ut tesaurum preciosissimum* (as a very precious treasure). It was lawful for teachers to board students, but severe penalties threatened them when they violated the duties of hospitality or lectured in another school.

Unlike the other European universities, the students of Bologna were distinguished by their maturity and prosperous circumstances. The elect youth of Europe convened in this city. It is estimated that no less than ten thousand German students were enrolled there between the years 1289 and 1796, at a time, namely, when such university institutions were not yet known abroad and teaching was imparted only to the wealthy who could allow themselves the luxury of a preceptor.

In the fifteenth century every Italian city had very flourishing superior institutes, while in northern Germany the first elementary schools were timidly beginning to appear. For this reason the flower of German nobility and talent, among whom the highest prelates, the greatest princes and literary men, drew from Italy and from Bologna the solid cultural foundations that kept alight the torch of their knowledge; deepest redress of the oppressed against the bloody arrogance of the conquerors.

Students grouped themselves in associations, each having a leader, according to their country of origin (*Universitates scholarium*). Citramontani (living this side of the Alps) were called the Italians, and Ultramontani (living beyond the Alps), the foreigners, who were subdivided by nationalities, each having their respective

Consiliarii (advisers). In turn, among these nationalities, a rector was elected, who had civil jurisdiction in controversies, conferred with the city magistrates, elected the lecturers and provided for their compensation and for school-books, which were purchased or rented from the *Stationarii* (stationers).

The audience was often so numerous in Bologna as to justify the order given to the lecturers in 1474, as they did in Siena, to assemble and debate in the public square. A statute of 1378 ordered *quod nullus possit legere de mane Medicinam nisi Bononiensis verus* (that no one but a thoroughbred citizen of Bologna could teach medicine in the morning), and this provision was obeyed until 1459, when it was revoked. Other ordinances prescribed the ordinary and the extraordinary lecture-books and hours, with the view of having the forenoon reserved for ordinary subjects. For the extraordinary lectures, given in the evening, only students could be employed who were duly chosen and salaried by the city, which, with the statute of 1410, assumed also the burden of the salary for the lecturers selected by the faculty college, provided they were of Bologna.

This is the first step toward State control and official recognition of the school, which, from a free and private institution, was thus changed into a dependent official organization. The last to receive proper legislation were the school buildings, an indispensable necessity for the teaching of medicine, if not for letters and law. Practical anatomy, at least, required special places for the keeping and dissection of the bodies. The task of providing for such a civil need was indeed not an easy one: ignorance, sentimentalism and religious fanaticism were jointly opposed to it. The earnest students, in retaliation, organized veritable punitive expeditions, snatching, carrying off, exhuming and mutilating bodies.

In the year 1727, in Bologna, a comic note was not wanting in this perplexing situation. Vesalius was represented contending with dogs and vultures which were devouring the remains of executed criminals at Montfaucon; Valsava was represented disinterring at nighttime horribly putrefied corpses; students were forced to return to relatives the bodies carried away or they were forced to take the bodies back to the places where they had found them, particularly if near to a church. Felix Plater dug graves with his hands; at funerals coffins were robbed of their contents; bodies were stolen from private residences; teachers were tried for the disinterment of an executed

criminal and consequent dissection of the body *cum rasuris et cultellis et alii artificiis* . . . (with razors, knives and other means). The chronicles of the time are full of such news, but it was not easy to find the parts of bodies carried away because they were hidden in private residences, where the courses in demonstrative anatomy were given. The lecturers vied with each other in forming their museums, the only place from which they could daily absorb school knowledge.

Where the intervention of governments was delayed the devotees of a particular science compensated this lack by founding an academy in almost every city. Thus was founded the "Cimento" in Florence, through the efforts of the de Medici's family; the "Pontaniana" in Naples; the "Platonica" in Florence; the "Cosentina" in Cosenza, the "Lincei" and the "Riva" in Rome, and the "Massari" in Bologna, contemporary of the "Philosophers" or "Traccia" of the same city. We owe to these private institutions the rending asunder from the mind of mankind the long night of ignorance and barbarism.

Subjects Taught. In the fifteenth century the subjects taught in Bologna were divided into two groups: those pertaining to law, and those pertaining to the art. Among the latter were included theoretical and practical medicine, surgery, moral and natural philosophy, astrology, logic, rhetoric and *Ars notaria* (notarial art), which, after the year 1357, was aggregated to the lawyers. A chair of Greek lectures was added in 1455, while that of rhetoric had, in 1439, already been divided into poetry, grammar and *lectura humanitatis,* which rose to such an importance in the fifteenth century as to attract many foreign students. The teaching of Hebrew, Chaldean, Arabian and of *De chronologia et de re diplomatica* (chronology and diplomacy) had short duration. Metrology and geometry were first included in astrology, and later, mathematics, algebra, mechanics and hydrometry were added. The chairs of physics and chemistry were established in 1737, that of optics in 1756 and that of agriculture in 1781. Since 1571 anatomy was taught separately from surgery, in which it had until then been included, and a few years later the teaching of *de simplicibus medicinalibus* was given a new title, *ad philosophiam naturalem* ordinariam (de fossilibus, de plantis, de animalibus). The chair *ad operationes chirurgicas* was instituted in 1733; that of lithotomy and ophthal-

mology in 1765; that of veterinary medicine and surgery in 1784, and, finally, that of *de variolarum inoculatione* in 1770.

Some of its Outstanding Teachers: Many of the lecturers promoted to the professorship, with the title of doctors, were foreigners and salaried by the commune or Gabel, and it is not difficult to imagine how they were antagonized by the native doctors, who claimed the exclusiveness in teaching.

The School of the Arts and, therefore, of Medicine flourished about two centuries later than the legists', that is, in the thirteenth century. It had very capable scholars in anatomy, among whom are particularly mentioned: Taddeo of Florence, whom Dante speaks of in the XII Canto of *Paradise*. His *Consilia medicinalia*, which in the total of 156 have been collected into the Vatican Code 2418, are still well known. Taddeo was the true founder of a school that had many followers, and drew from Boethius Latin doctrines. Dino del Garbo, who commented his Aphorisms, and his son, Tommaso del Garbo, who held the chair of surgery and published *Summa medicinalis*, were his pupils, as well as Guglielmo of Saliceto, the anatomist, clinician, sober reasoner, surgeon and author of *Summa conservationis et curationis* and of *Chirurgia*. Another pupil of Taddeo was Gentile of Foligno who published under the same title of *Consilia medicinalia* used by his teacher, a collection of practical observations. He also commented Galen's art, Hippocrates' and Avicenna's *Prognostics*, and wrote a book on the black plague, of which he was a victim in the practice of his duties.

A pupil of Taddeo was also Bartolomeo of Varignana, who continued the work *Consilia medicinalia* and wrote the *Quaestiones super libros Galeni de complexionibus* (questions on Galen's books about complexions). He was one of the most reputable physicians of the thirteenth century and the first one, perhaps, to obtain permission from the Senate of Bologna to dissect a human body publicly. The experimental trend established by him in anatomy found followers in the celebrated Mundino de Liuci, otherwise called Raimondo, and in Nicolo' Bertrucci Lombardo, both Taddeo's pupils who continued the work of their teacher. Mundino, too, wrote his *Consilia medicinalia ad varios morbos* (Medicinal advices for various diseases), and commentaries on Galen, besides a book on botany; his major work, however, is the *Anatomia*, which was adopted as text-book in all schools.

Taddeo's pupils were, also, the famous surgeon Lanfranco of

Milan, author of *Ars completa totius chirurgiae* (complete work on surgery), who taught in France, and Ugone Benci of Siena, who taught medicine in Bologna, Pavia, Parma, Florence, anatomy in Padova, and commented in *Consilia* Hippocrates, Galen and Avicenna's art.

Between the fourteenth and the fifteenth centuries we find Pietro di Azzolino of Argelata, to whom a statue has been erected in the anatomical theater of the Archigymnasium. He wrote six books full of observations on *Chirurgia* (surgery), and embalmed the body of Pope Alexander V, leaving with us a complete description of the process. Giovanni of Concorreggio, who also taught in Milan and Pavia, left us several interesting works: *Tractatus de febribus, Methodus medendi,* and *Summula de curis febrium.* We are told that Gabriele Gerbi, philosopher, anatomist and physician, of whom we know several works: *Cautelae medicorum, Quaestiones metaphisicae, Gerontocomia* and *Opus anatomiae,* was called to Constantinople to treat a Turkish gentleman of high rank and that, having cured him, was returning to his country when his patient relapsed and died. This gentleman's sons, suspecting his death due to administration of poison by Gerbi, overtook him at sea, sawed his accompanying son alive and inflicted the same penalty on him. His contemporaries were Alessandro Achillini of noble family, lecturer of anatomy in Padua and Bologna, author of treatises of anatomy, and describer of the parotid glands, cecum, choledochus duct, cerebral and spinal nerves, malleus and incus of the tympanum; and Jacopo Berengario of Carpi, Mondino's pupil, brought much honor upon Bologna school (1470–1530). This year is the four-hundredth anniversary of his death. He taught first in Pavia and, perhaps, also in Padua and Paris, then in Bologna, and dissected *plurima centena cadaverum* (more than one hundred bodies), as related by himself. Of his works the most famous are the *Commentaria* on Mondino's anatomy and the *Isagogae.* It is said that he, as well as Vesalius, Falloppio and Michelangelo, practised vivisection, an argument destitute of every foundation, as afterward demonstrated (see Roth in *Andreas Vesalius—vivisection des Menschen in sechzehnten Jahrhundert,* page 473, app. xi, Berlin, 1892).

Bartolomeo Maggi was held in great esteem. He was an archiater of Pope Julius III, surgeon and professor of anatomy, who published an interesting work on firearm wounds. Ulisse Aldrovandi, the prince of naturalists, a miracle of doctrine, of whom the University

of Bologna possesses the immense volumes on his anatomical studies upon animals, and his entire rich museum of natural history.

In the sixteenth century we have Volchero Poiter, a German, who for a short time was lecturer of anatomy in Bologna and published here and in Nuremberg two notable works; and then we have the most distinguished Andrea Vesalio, who taught in Padua, Bologna and Pisa, besides Paris and other European cities.

G. Cesare Aranzio, Maggi's nephew, lectured in Bologna from 1556 to 1588, and created the chair of ordinary anatomy, apart from surgery. He published *De humano foetu, De tumoribus, Observationes anatomicae,* etc., works on the human fetus, tumors, anatomical observations), remarkable for exactness of conceptions, especially in the part referring to intrauterine life.

His contemporary was Costanzo Varolio of Bologna, a clever professor of surgery and, later, of anatomy. In 1566 he was crowned with laurel and a statue in his honor was erected in the anatomical theater of the Archigymnasium. For his discoveries on the brain and the whole nervous system, the special senses, the larynx and the genitourinary organs he well deserves being considered one of the most valiant anatomists of his century.

Another remarkable man was Gaspare Tagliacozzo, in whose memory a statue was also erected in the Archigymnasium. He taught medicine and anatomy from 1570 to 1599 and published a work in two volumes of rare value. The esthetic and repair surgery (plastic surgery) was initiated by him. His contemporary and friend was Girolamo Mercuriale, whose vast culture was called "frightful." He taught from 1537 to the beginning of the year 1600, always to a large audience, in the schools of Padua, Bologna and Pisa, and was physician to Maximilian II of Germany and to Pope Gregorius XIII. He died in 1606, leaving many remarkable works on surgery; and this century was also illumined by the genius of Fabrizio d'Acquapendente and Fallopio of the School of Padua, and of Bartolomeo Eustachio of the University of Rome.

And the long list still continues. The end of the sixteenth century is honored by Girolamo Cardano, Gianfrancesco Rota and his son Flaminio, Antonio and Michele Sacchi, Francesco and Achille Muratori, and by Bartoletti, who left many books on medicine and anatomy, and, when still very young, died of the plague. The glorious list is closed by Gianbattista Cortesi, who taught anatomy in Messina and Bologna until his death in 1634. He contributed

many works on medicine and surgery, the most famous of which is *Miscellaneorum decades denae,* mentioned by Haller, published in a very limited edition.

In the seventeenth century the chairs of medicine passed to the Scotchman Georges Scharpes (Scarpius) and to the Irishman Neil Glacan, while Adriano Spigelio, professor at Padua's school, was rising, and with him Carlo Ruini, the keenest zoötomist of his time. When Harvey, then twenty, was studying anatomy in the University of Padua, Ruini published the *Anatomy of the Horse,* composed of five illustrated volumes, in which the study of the heart and of the circulatory movement of the blood was fully described.

A new era was at this time initiated with the creation of the *Accademia de Notomia* established by Bartolomeo Massari, afterward restricted to nine competent scholars designated by the name of *Coro Anatomico* (Choir of Anatomy). Among Massari's pupils are found Gianbattista Capponi, Carlo Fracassati and, above all, Marcelli Malpighi, who brought honor to the Bologna School and to Italy, of whom I had the pleasure of writing a modest biography in 1928 on the occasion of the three-hundredth anniversary of his birth, 1628. In his name and in his life is contained a monument of greatness whose size I might reduce in an attempt to summarize his complete work, and of Sbaraglia, an anatomist of the old school, his most obstinate antagonist; the mere mention of his name is even too much.

In 1697 the office of anatomical prosector was instituted and entrusted to Anton Maria Valsalva, Malpighi's pupil and teacher of the celebrated Morgagni of the Paduan School, a member of the Royal Society of London. An episode of his life is the disinterment of a body in advanced putrefaction, upon which the alteration of a nerve trunk had to be studied. He died of apoplexy at fifty-seven years, but will live forever because of his work, *De Aure Humana Tractatus* (on the human ear). He divided this work into two parts, anatomy and physiology, while his famous notes from which Morgagni drew his *Duodevigenti Epistolae Anatomicae* are an imposing testimony to the vast and profound critical doctrine of the compiler. His friend and associate in the investigations was Francesco Albertini, also of Malpighi's school, a modest and erudite clinician and anatomist, very sober in his writings: his *Commentario* (Commentary) is so dense with profound anatomical observations as to deserve its being placed among his greatest contemporaries. Other

celebrated scientific contributors were: Pietro Nanni, discoverer of the glandular follicles and of the pleural alterations, a famous physician, a prose writer and a poet; Matteo Bazzani, a philosopher, and Jacopo Pozzi, a poet, keen and expert observer; Ferdinando Guglielmini, who preceded Haller in the study of the function of the intercostal muscles; Gusmano Galeazzi, prosector, chemist, physicist, naturalist and geologist; G. Battista Stancari, chemist and keen student of the meninges; Giacinto Vogli, the author of *Anthropogonia;* Paola Balbi, physicist; Giovanni Molinelli, who studied the cerebral lobes and the decussations of the cranial nerves; Galli Bibbiena, biologist and naturalist; Giovanni Manzolini, surgeon and anatomist; Carlo Mondini, of whom we know the study of the appendix; Antonio Caldani, an ardent upholder of Haller; and, finally, Luigi Galvani, the famous demonstrator of animal electricity, who closed the cycle of the old lecturers and marked for the world with Volta, the beginning of a new civilization. Galvani was an anatomist and obstetrician, besides being physicist, chemist and meteorologist.

I would like to add here the names of two women—Laura Bassi, the teacher of Lazzaro Spallanzani, who held the chair of universal philosophy from 1732 to 1778, and that of Anna Morandi Manzolini, who was entrusted with the modeling of the anatomical preparations.

The Archigymnasium (The Original University Building). Among the most famous monuments of the period, the one still existing conserving the history of the glorious vicissitudes of Bologna's school, is the Archigymnasium Palace. It was erected through the indomitable will of Cardinal Pietro Donato Cesi, Bishop of Narni, when papal vice-legate governing in the name of the celebrated Carlo Borromeo (nephew of Pope Pius IV), who played a very important part in the resolutions of the Council of Trent. He was a very learned prelate, a scholar of the civil and canon rights, and deplored very much the fact that a school so famous throughout the civilized world had to occupy rooms in very modest houses and pay rent besides. He wanted everything united under one roof, and, as he knew how to find the necessary financial means, asking compulsory contributions even from students and professors, so he knew how to take personal control of the construction. Thus in a little more than one and a half years the Archigymnasium was erected, a sumptuous monument of architecture and one of the worthiest palaces of the period. The funds and

the plans which had been reserved for the construction of another building, Saint Petronius Church, were used for this purpose. As a result of this the church remains incomplete, a fact worthy of note when one considers that it was due to a prelate. The front of the Archigymnasium is marked by long portico; inside, next to the lobby, is a courtyard, austere in lines and, following it, the chapel, rich in ornaments and paintings, among which is the Annunciation of Calvart. On the sides of this oratory are the halls for the rectors, priors and counsellors, beautifully decorated with fresco paintings. Everywhere in the courtyard, corridors and on the walls of the stairways and meeting rooms there is a profusion of doctors' and pupils' blazons. On the first floor, above the chapel, is the anatomical theater, or dissecting room. We, accustomed to the naked simplicity of the modern anatomical schools, remain very much astonished at the sumptuousness of the seats and the wood carvings decorating the walls and the ceiling. A rich baldachin, supported by two life-size wooden figures, showing the first layer of muscles, indicates the lecturer's chair. Other life-size statues and busts of celebrated physicians and anatomists decorate the walls, mute witnesses for centuries of anatomical meetings, called functions, which were attended by the highest city authorities and a long retinue of ladies in great pomp. These functions lasted ten successive days while in the chapel below suffrage-masses were celebrated.

Another city solemnity was the preparation of the Theriaca, the universal panacea, that attracted the most learned and elegant public. It took place just in the center of the courtyard, the portico of which had been all decorated with damasks for the occasion. The ceremony was presided over by doctors and pharmacists in rich paludamenta fretting themselves about caldrons and stills, all intent in examining with devout scruple the ingredients used and the process followed, while the multitude of liveried servants and chevaliers in powdered wigs helped them.

At present in the upper halls of the Archigymnasium a library is located, and in every part of it long rows of legates', teachers' and pupils' coat-of-arms grace their vast walls: these were saved from destruction by the good sense of the people when, toward the end of the seventeenth century, the ill-intentioned republican revolutionary spirit of equality was raging.

But the living character of the past cannot always prevent the decadence of posterity: a strange phenomenon that falls fatally

upon the ancestors' greatness. Hence we witnessed in the seventeenth and eighteenth centuries, as I have already stated, a progressive decline in education which became infiltrated with the vain appearances that characterized the society of the seventeenth century, whence the sanctity of teaching was lessened by the weakness of the times. Professors, colleges and institutions deteriorated; pupils decreased in number and those few, whose youth did not favor meditation, abandoned themselves to every sort of unruliness and arrogance. The majority went to other universities that were thriving in every place. The phenomenon became so serious that at the end of the eighteenth century there were scarcely ninety lecturers for less than twice as many students, who, to their boldness, added sarcasm, thus creating the comic character of Dr. Ballanzon, worthy expression of the presumptuous aspect of the empty teaching of the age. The entrance of the French into the city and the change of government produced, even among the students, a sense of rebellion against the old pontifical régime. The University corporations were abolished, privileges destroyed and the coat-of-arms removed. The Archigymnasium became the seat of the constitutional circle and, afterward completely abandoned, ran the risk of being transformed into a synagogue. It was, however, used for the elementary school for thirty years, that is until 1838, when it was occupied by the library.

When we observe today the serious self-possession of the students, moved by respect among those imposing walls, every corner of which is a book of history, we can hardly understand how the very same places could have become the stage of impertinent, arrogant students; and we are inclined to meditate on the fatal course of those centuries when laxity of customs and of constituted powers, a natural reaction against tyranny, remained arbiter of history. The thousands of students now annually congregating in the University of Bologna, again under a free régime, pass by almost unconscious of the history of the austere building. They focus their attention on the courtyard. There they may catch a glimpse of the balcony, on the railing of which a frog disclosed to Galvani the mystery of animal electricity. They now move on to the city limit, where the Temples of Science are located. Sadly the Archigymnasium remains, in its vast halls, solemn guardian of an endless tradition of treasure of precious incunabula, richly miniated, surrounded by the manuscripts of our great leaders.

APPENDIX

INTRODUCTORY NOTE TO THE SECTIONS

ALMOST all of the papers read before the various sections of the College are subsequently published in full in medical periodicals.

The papers listed in the following pages may be found by consulting the *Index Medicus,* provided a sufficient length of time has elapsed from the date of publication to the next succeeding quarterly number of the *Index Medicus.*—[EDITOR.]

PROCEEDINGS

OF THE

SECTION ON OPHTHALMOLOGY

JANUARY, 1930 TO DECEMBER, 1930

JANUARY 16

Iridectomy in Bullous Keratitis

Dr. William Zentmayer presented a case of relapsing bullous keratitis in a man, aged sixty-eight years. The condition was of about one year's duration, when an iridectomy was done which has apparently resulted in a cure, as no recurrences have taken place in eight months and the eye is functioning normally with a visual acuity of 6/15.

The patient has been constant and faithful in attention to the eye, having been regularly in attendance at one clinic for six months, where the usual procedures, including radiation therapy and the thermophore, were used without benefit. The etiology was obscure. The only possible contributing cause was an ethmoiditis, for which an operation had been successfully done early in the eye disturbance. Aside from the relapsing bullæ with the accompanying severe subjective symptoms, temporary slight increase in tension and an alteration in the anterior lens capsule, the eye was normal.

Discussion. Dr. Cowan said he had seen this man quite a while before Dr. Zentmayer, at the Wills Hospital, and was very much interested because he presented the same condition as that which he and Dr. Holloway had shown some time before at the Section. There was a bluish-white, punctate deposit on the anterior surface of the lens, forming a sort of lacework design. The ragged edges extend forward into the anterior chamber. At present there is a difference of opinion as to the etiology of these cases.

The same type of case is illustrated by Bedell in the Jackson anniversary book. He called it a proliferation of the superficial layers of the lens capsule. Vogt, in his *Atlas*, illustrates the same condition and considers it a peculiar form of retained pupillary membrane. He pointed out that

the design roughly outlines the arrangement of vessels of the tunica vasculosa lentis. Dr. Holloway and Dr. Cowan, agreeing with Vogt, presented their case as one of persistent pupillary membrane. Since then, Vogt has reported several other cases of the same kind, but now considers the exudate due to a degeneration of the superficial lens capsule. Meesman, in his *Atlas*, illustrates the same condition and believes as does Vogt. Kraupa includes these cases with glassblower's cataract and believes that the exudate is due to a glandular degeneration of the capsule lamella. Sehick believes that the exudate is a deposit of cells from the posterior surface of the iris.

The first case that Dr. Cowan saw was one of Dr. Ring's. Shortly after this the case was seen at the University, and then the case shown tonight, all within a very short period of time. He thinks these cases are much more common than is supposed. They always occur in elderly people, with other degenerative changes and very often with glaucoma. The case of Dr. Holloway and Dr. Cowan developed glaucoma a very few days after he was shown.

Dr. Griscom said that some years ago he reported a case of bullous keratitis before the Pennsylvania State Medical Society, in which the history was quite similar to Dr. Zentmayer's. The patient was a lady, aged about sixty years, who came to the Wills Hospital with recurrent bullæ of the cornea, which repeatedly ruptured and reformed, accompanied by intense pain. There was a rise of tension in this case, but no other evidence of ocular disease aside from the bullæ.

All efforts to relieve the patient, including iridectomy, were of no avail, and it was finally decided that the eye should be enucleated because of the intense pain. In the course of the examinations prior to this operation it was discovered that she had an active ethmoiditis. This was treated and, whether because of this treatment or not, the bullæ ceased to form and the eye remained quiet.

Dr. Griscom referred to the above history because, after one and a half years, this patient returned to the hospital with a similar condition in the other eye. In spite of all treatment, including treatment of the ethmoids, which were not actively diseased at that time, she continued to have repeated attacks. The patient finally became discouraged and disappeared, but it is quite likely that someone has since enucleated the eye.

Dr. deSchweinitz, after congratulating Dr. Zentmayer on the admirable operative result he had achieved, related briefly the case history of a young Hebrew man, who, long the subject of relapsing bullous keratitis which had resisted all the usual nonoperative procedures, was finally cured when, an unusually large bulla having formed, all the loosened epithelial layer was removed and the exposed surface painted with a 1 to 20 solution of trichloracetic acid, especially applied along its edges.

No definite cause for the formation of these large bullæ was discovered. The patient was highly myopic, had dwelt in a malarial region and had frequently received full doses of quinin, but prior to the cauterization described these had had no salutory influence on the keratitis. The slight corneal haze which remained caused no noteworthy visual inconvenience.

In closing, Dr. Zentmayer said that Dr. Buchanan had brought up the question of malaria as a cause of the bullous keratitis. The patient stated that, many years ago, he had a "touch of malaria," but this seemed too long ago to have any bearing on the etiology of the corneal condition.

A Case of Electric Cataract

Dr. Holloway presented the history of a case of electric cataract. The essential points were as follows: A boy was subjected to a shock from an electric wire carrying 2200 volts. As far as could be ascertained, inflammatory signs had never been present. Failure of vision was first noted nineteen months after the injury, and when the patient was first seen by Dr. Holloway, ten months later, or twenty-nine months after the accident, there was complete opacification of each lens. There was a small, central, capsular placque on each cataract.

Following iridectomy and evacuation of the lens, the patient had 6/12 vision with the right eye and 6/6 with the left. No demonstrable fundus lesions were present in either eye.

Dr. Holloway (in closing) said that he had intentionally avoided saying anything about the pathogenesis of this type of cataract, as he hoped some of the men interested in biochemistry and pathology would discuss this point. As far as he knew, it was probable that the observations of Hess were those generally accepted. This observer had found that, subsequent to an electric discharge, there occurred a distortion of the capsular epithelium with the accumulation of fluid between the capsule and the lens fibers.

Adenocarcinoma of Conjunctiva

Dr. Edward B. Spaeth reported a case in which the growth was at least of one year's duration, and in the past few weeks was showing a very rapid increase in its size. The greatest part of its present size had been reached in the past three weeks. The tumor mass at first examination extended from the limbus, where it had grown over upon the cornea for about 2 mm., toward the outer canthus for a bit more than three-fourths of the scleral surface, and in width it was slightly more than that of the normal palpebral fissure. It was elevated at its highest point for several millimeters, multilobed, soft and with a very copious blood supply. It was firmly adherent to the underlying sclera.

The limbus is the most common site for epibulbar carcinomata. In this case there is, therefore, nothing unusual as to position. Also, epibulbar carcinomata, as a rule, respond rather readily to radium treatment. They are rather rapid in their growth. Adherence to the underlying tissues occurs late in their course and they will usually extend completely about the limbus before penetration occurs, because of the resistant sclera. In this case there are a few points of difference from these usual features. These are: its very slow growth, its penetration to the underlying tissues, its very copious blood supply, the brown granules of pigment scattered throughout the tumor mass and its unusual resistance to radium therapy. Radium was applied by conjunctival contact three times at two-week intervals, with but little change in size or its appearance. Ten days after the third application it was resected from the sclera by actual cautery. A fourth radium application was done the same day. The case then went on to uneventful recovery and has remained so for six months.

Judging from the age of the case, with its rapid growth, it is possible that this started originally with an epithelial plaque, a benign adenoma starting from glands of a rudimentary acinotubular character or from a nonpigmented mole. The late sudden growth would indicate the time at which malignancy developed therein. This is also consistent with the pathological report made upon the sections.

An incipient cataract has remained unchanged throughout the course of this treatment. Vision is still 6/5 with correction.

A Method of Uncovering Latent Hyperphoria

Dr. Leighton F. Appleman presented a method of uncovering latent hyperphoria which consists essentially of three steps: *First*, with a Maddox rod the muscle balance is tested at a distance of 6 meters. This reveals the manifest error at this distance. If a deviation from the normal is present, a prism is placed before the eye in such a manner as to obtain equilibrium. *Second*, the patient is given a card on which is the dot-and-line of Graefe. He is told to hold this at the reading point with the line horizontal in testing for hyperphoria. A 20-degree prism is then held in front of one eye, with its base toward the nose. Lateral diplopia is thus produced, and, if no hyperphoria is present, the images will be on the same

horizontal plane. Should one be higher, a prism of sufficient strength is placed before one eye to bring the images upon the same horizontal plane. *Third,* leaving this prism on, the patient is told to look at the point of light 6 meters distant, or across the room. The Maddox rod is again used and, if the patient now shows an overcorrection, the strength of the prism is reduced by half degrees until the eyes are again in equilibrium. This constitutes the second distance estimation. The difference between this and the amount shown by the first test constitutes the amount of latent deviation.

In an analysis of 500 cases there were 253 in which primarily no hyperphoria was shown, but in which from 0.75 degree to 3 degrees were uncovered by means of this method of testing. In the remainder, while a low degree of deviation was found at the first test, considerably more was revealed as latent.

Incorporation of these findings in the lenses for constant wear produced gratifying relief of symptoms for which the patients sought relief.

Discussion. Dr. Zentmayer said the objection that came to his mind was to the high prism used. The slightest angulation of the prism may produce this apparently latent hyperphoria, but that he supposed care was taken to see that this did not occur.

Although he did not wish to put his opinion above that of Mr. Maddox, supported by Dr. Appleman, that the correction of hyperphoria reduces the amount of lateral deviation, in his experience it had no such effect. In his opinion, the correction of hyperphoria under 2 degrees is rarely necessary. He considered that where the refraction work was thoroughly done it was seldom necessary to give special attention to the correction of a low-degree hyperphoria.

Dr. deSchweinitz said he was entirely convinced that the prismatic correction of hyperphoria represented a highly important therapeutic procedure, and often, moreover, when the hyperphoria was of small degree and the most accurate correction of the refractive error had failed to achieve the desired result.

In his experience and that of his associate, Dr. Fewell, when the hyperphoria is latent, the Marlow method of uncovering is a most satisfactory one. Having had no experience with Dr. Appleman's technic, he was unable to discuss it in relation to that advanced by Marlow.

He referred to the fact, historically interesting, that the late Dr. E. O. Shakespeare had corrected hyperphorias with prisms at a period long antedating the well-known studies of the late Dr. George Stevens. He was not sure, however, whether Dr. Shakespeare had placed his cases on record.

Dr. Holloway said that he had used Dr. Appleman's routine in several instances and found that his results were similar to those obtained by Dr. Appleman. As far as a prism was concerned, it was not necessary to use as high a prism as has been suggested. He felt that the test was essentially a fogging of the muscle, in the same sense that this word is used in a refraction case. He thought that all clinicians obtained satisfactory results from the use of vertical prisms, but occasionally one obtained just as satisfactory a result from taking off a prism that had been previously prescribed.

He considered the Marlow test a distinct acquisition to the methods of treatment for muscle imbalance, and he generally used it for several days. One of the most grateful patients he had was one who had been wearing a vertical prism placed in the wrong direction, the error being uncovered by the Marlow test.

Dr. Fewell said, in regard to the Marlow test, that patients do object to wearing a bandage over an eye, but that in patients who are already wearing glasses, by making one glass frosted, he had seen $\frac{1}{4}$ degree of hyperphoria change to a 1 or 2 degree after wearing this for one or two days. Patients will not object as much to this frosted glass as they would to the bandage.

Dr. Langdon said that in the case which Dr. deSchweinitz referred to as having seen in consultation with him, a frosted glass was tried for a week, and for the next week a tight bandage over the eye was worn constantly during the waking hours. The patient found the bandage much more comfortable and less annoying than the frosted glass, and incidently, a hyperphoria of $3\frac{1}{2}$ degrees was developed, where less than 1 degree was noted previously.

Dr. Carpenter said that he at least had had the privilege of looking back over a good many years of ophthalmic practice, and that he was rather astonished to find that agreement had not been reached as to the prism for vertical muscle deviation. He believed that every eye, properly examined, which showed a relatively high defect should be corrected by vertical prisms, even after a most perfect correction of the refractive error.

He said that there are some people who have such a splendid nervous system that they can go with hyperphoria, or hyperopic astigmatism, with glasses 15 degrees or more of faulty placement of the cylinder and come back and sing the doctor's praises. He had met one of these cases who had 5 degrees left hyperphoria. He ordered prisms $2\frac{1}{2}$ degrees, base up, for the right eye, and $2\frac{1}{2}$ degrees, base down, for the left. The patient was a very busy lawyer and did not return to him for two weeks, when he found that the prism $2\frac{1}{2}$ degrees, base down, was placed base up, so that he was wearing 5 degrees in the wrong direction and was delighted with the result.

Dr. Carpenter felt that hyperphoria is most important and should be treated, and he did not see why the Maddox test could not be used for the 13-inch test exactly as in the 6-meter test.

Dr. Appleman (in closing) said that he thought the reason there was not more unity of opinion as to the value of prescribing vertical prisms was that the majority of examiners have not a definite method for uncovering

these errors. While there are many different methods of estimating the muscle balance, and while it is probably true that some will get results by using one method or another, he felt that everyone should stick to some one method by which these latent defects may be uncovered and corrected as part of each examination.

He stated that he had been led to a recognition of the importance of correcting low degrees of hyperphoria in the course of the examination of many medical students who, at a first examination, took a certain correction and wore it comfortably for a year; at this time they began again to have discomfort and, upon examination under exactly similar conditions, showed exactly similar refractive errors as found the year previously, excepting that a low degree of hyperphoria was uncovered. The prescribing of a vertical prism as low as 0.5 degree has enabled them to go on with the second year's work with perfect comfort.

In regard to the lateral deviations, it has been his experience that, aside from the need for convergence training by means of prism exercises, the lateral deviation usually takes care of itself.

In regard to the strength of prism used to obtain lateral diplopia in testing at the near point, he mentioned that he used a 20-degree prism. It is not necessary to use one this strong provided that diplopia is produced, but it is with this one that he has been accustomed to work.

FEBRUARY 20

Dr. Burton Chance exhibited a case of a man whose four eyelids he had restored to usefulness by skin graftings. The man had been in a railway wreck. His body being caught under the locomotive, the fire and the escaping steam had burned his face, head and neck severely. When first seen by Dr. Chance the corneas were ulcerous, and all four lids everted by cicatrization. The globes are now protected, the corneas healed, the cicatrices on them fading, and the man has useful sight, so that he is able to perform yard duty.

Discussion. Dr. Griscom said that about four years ago he had shown a case before the Section somewhat similar to the one exhibited tonight by Dr. Chance.

Following an extensive burn of the face, due to a gas-flame, the patient had a bilateral cicatricial ectropion of both lids. The operation consisted of freeing the lids by dissection and performing a tarsarrophy, after which a large Thiersch graft was placed over the denuded area and a firm bandage applied.

The ultimate results were entirely satisfactory and the patient had almost complete restoration of normal lid function.

Glioma

Dr. Burton Chance said that the seriousness of diagnosis in all cases of glioma and the importance of thorough histologic study of the tissues after excision of the globe, in any case, compelled him to again call attention to the case of the boy, aged eleven years, that he had brought to the meeting last November.

It was recalled that in the left eye of a healthy, vigorous Italian boy, a whitish fungus mass, which filled a large portion of the vitreous chamber without provoking inflammatory symptoms, was found. The tension of the globe was not elevated. The disease had become manifest only a month or so previous. As the case was studied in November, there was some doubt as to the diagnosis. It seemed inflammatory from one angle and gliomatous at another. The consensus of opinion, however, pointed to glioma. The boy went home over Thanksgiving. On his return the Saturday after it was seen that the mass had grown larger and the eye was painful. Enucleation was performed and the globe sectioned. The mass resembled a collection of inflammatory exudate. In December it was reported that the only cells found in the many sections studied were of an inflammatory nature. Dr. DeLong and Dr. Chance were not satisfied with these findings, so the remainder of the block was cut and studied, with the result that in the portion in the lower nasal quadrant of the globe there was found a mass of true neuroblastomatous cells in the retinal tissue. Neither the nerve nor the choroid had been involved. The aberrant cells are occupied by a very large nucleus.

Dr. Chance said such a case as this presented confusing characteristics. When the transilluminator was applied there were no shadows, but could there have been when the mass was on the posterior wall of the globe?

Cholesterin in the Anterior Chamber

Dr. Leighton F. Appleman showed a case who had come under observation at the Wills Hospital on February 1, 1930, because of poor vision in the left eye, his best eye. His right eye was blind, due to the formation of a cataract within a month following a blow upon this eye fourteen years previously. Inspection of this eye revealed the cornea clear; anterior chamber slightly deeper than normal; pupil round, 4.5 mm.; slightly dilated, no reaction to direct

light, but indirect reaction prompt; lens remains clearly visible in the pupillary space. No light perception. No irritation of the eyeball.

Vision in his left eye, he stated, had become hazy about ten years ago while at work, and inside of twenty-four hours the sight was reduced until it was little better than it is today. For this he was treated in the hospital of the University of Virginia, being sweated by means of hot packs, in addition to other medication. Under this treatment the eye improved and seemed all right for six years; but then, four years ago, it began to fail, until it reached the condition now seen. The condition of this eye has not changed since he was first seen on February 1, the cornea being clear; the anterior chamber of normal depth; the pupil round, semidilated, reacts to direct light, but no indirect reaction present; the iris has a bluish color in contradistinction to a brownish color of the right.

With the ophthalmoscope, no view of the right fundus was possible. In the left eye, a practically normal reflex was seen through the lens, which showed opacities beneath both the anterior and posterior capsules, while the center remain uninvolved. The vitreous was, and still is, cloudy, fluid and contains abundant opacities, many of them dark in color, stringy and containing uveal pigment, as can be readily seen with the slitlamp. The disk and vessels cannot be made out, nor any details of the periphery. Vision reduced to counting fingers at 6 inches, with light projection good.

On February 11, 1930, in leaning over, he struck the right eye against a chair. On February 13, he came again to the clinic with the statement that, following this blow, the eye was painful. It was injected around the cornea, the cornea itself was clear, the anterior chamber slightly cloudy and containing a few cholesterin crystals. No fresh blood was present. He was taken into the hospital and local treatment given.

The accumulation of cholesterin crystals increased until they practically filled the anterior chamber, together with an exudate or deposit, which looked like lens material. It now seems to be settling to the bottom of the chamber.

With the sliplamp, it can be seen that the zonule is broken on the temporal side and the lens displaced inward and slightly downward. The iris, too, is seen to be greatly congested, and numerous small vessels can be seen over its surface.

It is rare that cholesterin crystals appear in the anterior chamber.

In this case I believe they come from the vitreous, as the result of a break in the zonule following the recent injury. Their presence in the vitreous previously can be assumed to have occurred as the result of degenerative changes following the injury fourteen years ago.

The case is brought before you as an example of a rather unusual condition. It will be interesting to see whether these crystals absorb or whether shrinkage or other changes supervene. At this time, nine days after the injury, the cholesterin crystals seem to be settling to the bottom, as the upper half of the anterior chamber is much clearer than two days ago.

Discussion. Dr. Zentmayer said he had seen at the Wills Hospital a case of iridocyclitis in which there were large masses of cholesterin crystals in the anterior chamber. The chamber was washed out with salt solution, but refilled with cholesterin. The condition continued to recur and the eye was ultimately enucleated.

Because of the recurrence of the condition it was thought at the time that they probably found their way from the vitreous into the anterior chamber. The position of the lens and the condition of the zonule could not be determined.

Dr. Chance said that some years ago, when Dr. Koeppe was giving a course of lectures at the Wills Hospital, he brought a patient who had had an accident, and with the corneal microscope there was demonstrated the presence of these crystals. Dr. Chance had never seen a case similar to the one shown tonight, wherein so many cholesterin crystals floated in the aqueous.

Dr. Cowan said that he had seen this case at the Wills Hospital and had examined it with the slitlamp. He had never before seen a case of this kind. The aqueous was slightly cloudy and the anterior chamber filled with brilliant red, green and silver crystals.

Exhibition of Case of Heterochromic Cyclitis

Dr. Warren S. Reese brought a case before the Section which he thought might be of especial interest in view of Gradle's recent paper. A young lady, aged twenty-one years, had consulted him last November for a routine eye examination. The external examination at that time, as at present, was negative except for the heterochromia. Her mother is certain that her eyes were of the same color as a child. The change in color appeared around the age of twelve, when she was examined by an optometrist, whose meager record was secured.

Ophthalmoscopy revealed a descemititis, beginning cataractous changes and numerous vitreous opacities. Vision was improved to 18/30. Both eyes showed a compound hyperopia, but the error was small in the unaffected eye, whereas the left or affected eye required a rather high cylinder. The optometrist found a somewhat similar change, so that presumably this difference was congenital. The cause of this affection would appear to be the teeth.

There are several interesting phases of this condition. It should be understood that there are two main types of heterochromia, the congenital and the acquired, or that due to some pathologic change as in the present case. Fuchs is somewhat ambiguous on this point. In one place he states that the lighter-colored eye is predisposed to cyclitis; but, later on, he cites cases in which the cyclitis preceded the heterochromia. Gradle states that most authors consider the heterochromia the result rather than the cause of the cyclitis or inflammatory condition. The extreme chronicity of these cases is very remarkable; likewise the absence of inflammatory symptoms and posterior synechiæ. The use of a mydriatic may be a perplexing question; none was used in this case until recently, when the eye became painful. This was relieved by homatropin. Dr. Cowan made a slitlamp examination.

Discussion. Dr. Cowan said that with the slitlamp this is the usual picture of heterochromic cyclitis: Precipitates on the posterior surface of the cornea; specks in the aqueous; deposits in the pupillary space; changes in the iris and in the lens. In the right eye are a few pigment deposits, such as are often found and considered by some to be remains of pupillary membrane. There are a few of the same kind in the left eye; but here, besides the pigment deposits, there is an almost complete meshwork of exudate on the anterior surface of the lens. There are many atrophic areas in the iris, especially around the pupillary margin. The changes in the posterior portion of the lens lie in front of the capsule in the cortex; those in the anterior portion consist of opacities and vacuoles under the capsule. This is the paler of the two eyes.

Dr. Cowan said that, while it may be true in cases of heterochromic cyclitis, a pale iris preceded the cyclitic and lenticular changes, he felt certain that after a cyclitis in any eye there is often loss of pigmentation of the iris. In a case of such long standing as this we should expect the iris to be lighter than that of the healthy eye. Pigment migrates and is carried onto the lens surface and is also deposited on the posterior surface of the cornea.

Dr. Zentmayer said his interest in heterochromia irides had recently been renewed by seeing a woman, aged forty-four years, with this condition associated with Raynaud's disease, of the first degree.

It is difficult to know whether the eye condition has been acquired or whether it is congenital. Notes taken by former ophthalmologists make no mention of a difference in the color of the irides. The right iris is of a blue-gray color and the left a hazel. Right pupil is 4.5 mm., left pupil, 3.5 mm. In the right eye there are precipitates on the posterior surface of the cornea. With the slitlamp they appear a light gray in color, with crenated edges, and are fairly evenly distributed. There are a few fine vitreous opacities. The iris is partially atrophic, the stroma has a somewhat fibrous appearance and the crypts are almost eliminated.

The vision in the right eye is 6/6 and in the left it is 6/5. The tension is 18 in the right and 22 in the left eye. The refraction is a compound hyperopic astigmatism in each eye.

Aside from Raynaud's disease, the physical examination is negative. There were no indications of tuberculosis or lues.

The association of local asphyxia may be more than a coincidence. There are on record at least 2 cases of heterochromia irides with a Horner syndrome, and the cause of Raynaud's disease is a sympathetic disturbance. Of course, if there is a sympathetic element in these cases it would be paralytic in Raynaud's and Horner's diseases. Gradle, in his recent paper on heterochromia irides, asks the pertinent question why these cases with a moderate degree of cyclitis, which he thinks to be probably of tuberculous origin, almost invariably develop cataract, while this does not occur, as a rule, in frank cyclitis of similar origin, with exudate lasting over a long period. In his series of 13 cases the only evidence of tuberculosis, however, was a positive von Pirquet in 3 cases.

Dr. Chance said he was very much interested in the classification which Dr. Reese has given. In the first ten years of practice he had hunted out congenital malformations. In that time he saw many cases of heterochromic irises and had followed many of them, but could not recall any which later became inflamed. He would, therefore, consider all such as congenital types. He had, however, seen numerous instances after iritis where discoloration had occurred, yet he doubted if many of the congenital type became inflammatory. He has known a young lady, aged twenty-six or twenty-eight years, all her life; the iris of one eye is half gray and half brown, and she has never had any inflammatory signs in her eye.

Dr. Langdon said the change in color of the iris which comes on after iritis, and which is due to congestion and exudation into the iris, is very different from the change in color which comes on from degenerative processes where there is atrophy and where it is due to pigmentation.

It seems to him that this is the condition in the present case of Dr. Reese.

Hole at the Macula

Dr. George H. Cross said that this condition was brought to our attention, in 1869, by Dr. H. Knapp, and by Dr. H. D. Hoyes, in 1871, and more thoroughly studied by Kuhnt and Haab, in 1900. It was after this date that these cases were reported more freely:

E. Fuchs (1901), deSchweinitz (1904), E. von Hipple (1906), G. Coats (1908), Tweitmeyer (1909), Zentmayer (1909), R. F. Moore (1910), Clapp (1913), Burkholder (1918), Sedgwick (1918), Maxey (1919), Cross (1919), Lister (1924), Scarlett (1925), Bedell (1926).

Holes at the macula may be divided into two general groups: traumatic and nontraumatic.

As the case I wish to present this evening was produced by traumatism, my remarks will be confined to cases in this category.

During the war, while stationed at Camp Meade, we collected from 4 to 6 cases, all in men who gave a history of accidents occurring in civil life. One case, that of a colored man, stated he had been hit on the head with a baseball bat.

Following the war, at the U. S. A. General Hospital No. 11, Cape May, we had a number of cases of hole at the macula, following the traumatism of war injuries. One very extraordinary case was published in the Government Medical History. This young white man, aged twenty-two years, was struck by a M. G. B., which entered the malar bone 3 cm. below the external canthus of the left eye, directed across the face, and emerged below the right eye at the inner canthus. Photograph exhibited, which shows the hole at the macula, four ruptures of the choroid, numerous cholesterin crystals and a large area of choroidal disturbance.

The second case was that of a young white man, aged eighteen years, who was wounded in France, August 11, 1918. He had three large, depressed scars, with loss of bony substance, in right frontal region. In the right eye he had an oval hole in the macula, deeply pigmented and with cholesterin crystals present. There is some retinochoroidal pigmentation along a branch of the upper temporal artery. A water color of this case has been made.

These two cases and the case shown at the Section were white, while the cases described by Burkholder, Maxey and Sedwick occurred in colored men.

The case shown at the Section was a boy, aged fifteen years, struck in the left eye with a football in the summer of 1928. Following this injury the hole in the macula developed. This hole is of the typical, slightly horizontal type with numerous cholesterin crystals seen. There is a marked retinochoroidal disturbance. His vision in this eye is 20/100.

The pathology of macular hole, as studied by Fuchs, Coates, Von Hipple and others, seems to point to a cystic degeneration in

the retinal layers, with subsequent rupture and formation of a hole. Coates thinks that the degeneration is due to an edema of the retina at the posterior pole, involving usually all of the layers of the retina, but may produce a retinal hole from edema of the inner layers only.

Fuchs points out that the pathology may be the same whether due to traumatic or inflammatory changes. There may be cystic formation followed by pressure, atrophy or rupture and hole formation. He also considers that a contusion may result in serious molecular changes and death of the cells, with absorption without edema or cystic formation.

Discussion. Dr. Zentmayer said he had seen quite a number of cases of hole at the macula. One interesting case he recalled was the result of edema from sclerosis of the macular branch of the superior temporal artery in an old lady. The localized edema had caused a typical hole in the macula about one-third the diameter of the disk. Nearly all these cases have a very fine pigment rim the result of turning out of the pigment layer of the retina.

Dr. Chance said 8 cases were referred to him at the Army hospital in Cape May. He recalls one in which there was no actual wounding of the eye; there had been an explosion a great distance away. There had been an immediate laceration of the retina from the effects of an "air blow." Dr. Burkholder had illustrated a similar case.

He thought it might be of interest to the Section to hear of two early records of this type of case. In going over the records of the late Dr. Thomson he was attracted by a very complete sketch which Dr. Thomson had made in the early 70's. It was the case of a young woman who had been struck in the eye by a rubber ball filled with water. Dr. Thomson apparently thought he was dealing with a congenital condition and did not associate it with the accident. The second case was recorded in 1887, of which careful drawings were made, a young lady struck in the eye with a half of a lemon. These cases were probably not reported in the literature.

Dr. Cross said that many cases of so-called holes in the macula are really not holes in the macula. He had been asked to see a case a few days previously and found it was simply a case of central macular choroiditis, and he thought it possible that other cases of macular choroiditis had been reported as holes at the macula.

Treatment of Vernal Conjunctivitis by Radium

Dr. Ronald C. Moore (by invitation) reported the case of a boy, aged twelve years, who has had vernal conjunctivitis for two years. He reported first to the Eye Dispensary at the Presbyterian Hospital in May, 1929. Radium treatments were started in October,

1929—two treatments to the right lid and one to the left. The boy is now symptom-free.

Vernal conjunctivitis was first noted by Arlt, in 1846, in his paper entitled, "Three Cases of Infiltration of the Limbus by Grayish-yellow Transparent Gelatinous Masses."

The disease is of rather rare occurrence—1 case in every 200 to 500 cases of conjunctivitis. Males are affected more frequently than females, in the proportion of 85 to 15 per cent. It runs a protracted course of from six to ten years, and in some cases as long as twenty years.

There are three varieties of vernal conjunctivitis: (1) Limbus; (2) palpebral; (3) mixed form. Usually bilateral, although unilateral cases have been reported.

The diagnosis is the most important factor, as the disease is to be differentiated from trachoma, chronic catarrhal conjunctivitis, phlyctenular conjunctivitis and the conjunctivitis of hayfever.

The granulations in vernal conjunctivitis are flatter than in trachoma, although in some cases, where the granulations are round and pinkish, yellow differentiation is more difficult. The history of the case and absence of vascularization of the cornea and secretion will usually exclude trachoma. According to Elshnig, normal vascularization in vernal conjunctivitis is replaced by great numbers of small bloodvessels, rising perpendicular to the conjunctival surface. The nodules of eczematous conjunctivitis break down speedily, while those of vernal conjunctivitis never break down and may last for years.

X-rays have been used, but radium can be more accurately dosed; therefore, it is the best remedy known at present. If too great a reaction is produced, cataract, glaucoma, atrophy of the iris or destruction of the eye may result.

In conclusion, it may be stated that vernal conjunctivitis can be relieved with radium in the proper dose in all except the limbal form, where danger of damage to the globe prohibits the use of radium. Radium in proper doses has been very favorably reported on by most authorities.

Discussion. Dr. Shumway said that the patient to which Dr. Moore had referred was reported before the Section in 1918. Originally seen in 1913, he had been treated with 11 or 12 mg. of radium, which was all that was available at the time, but without success. Subsequently, in 1918, he had been treated with doses of 35 to 50 mg. by Dr. Pancoast, the lid

being everted and the filtered radium applied close to the conjunctival surface. The thick lid masses disappeared permanently. He had reported 3 other cases before the Section on Pediatrics of the State Medical Association in 1918, also with cure of the condition. One case was a private one, and had been seen frequently since then. The conjunctiva was smooth but atrophic and had a tendency to form a skinlike epithelial coating which had to be rubbed off. The danger of the method lay in the loss of eyelashes, which usually returned, but not always. The lid masses were more than thickened epithelium, true fibrous tissue being formed, with much elastic tissue, and ordinary treatment had no effect on them. He had never had any success from the use of fibrolysin.

Dr. Cross said he had brought with him a boy, about seventeen years of age, with a typical case of vernal conjunctivitis, who had had three applications of radium by Dr. Pendergrass at the University Hospital, merely for the sake of comparison with the case on the program in which the treatment had been successfully carried out.

He followed out this line of treatment because of the good results in a similar case which he had a number of years ago.

Dr. Louis Lehrfeld said that, through the courtesy of the staff of the Wills Hospital, he had resumed his studies of vernal conjunctivitis, which began in the clinic of Dr. Burton Chance in 1924.

Last year the studies were confined principally to the various therapeutic measures commonly used in the treatment and cure of vernal conjunctivitis. Among these therapeutic measures considered was the use of radium. In the series of 18 cases referred to him last year, 11 were treated by radium.

In coöperation with Dr. Widmann and Dr. Spackman, he was planning to so standardize the use of radium element in the treatment of vernal conjunctivitis, that it might be used uniformly by all radiologists and ophthalmologists with safety and with definite results.

Dr. Lehrfeld said that, inasmuch as radium has been heralded as a specific remedy for vernal conjunctivitis, this particular measure of treatment was selected with a view of determining definitely its efficacy and manner of application.

In lieu of the possible dangers to the eye by the use of unfiltered radium, treatment was begun in a series of 11 cases, using 50 mg. of radium element, filtered by a brass capsule 1 mm. in thickness, and by rubber 2 mm. in thickness.

The application was made directly through the closed eyelids, so that the entire conjunctiva and the cornea might receive the benefits of the radiation. This was thought to be an advantage over the application to the inverted eyelids, because it is impossible to get uniform application to all parts of the eye with the eyelids turned up.

Since vernal conjunctivitis may affect all parts of the conjunctiva and not the lids alone, it was felt that the application through the closed eyelids, with the protection of the eyelashes with a plate of lead, is advantageous.

The application is made for a period of one-half hour, the radium being exactly 3 mm. from the skin of the eyelid, and there is delivered 25 mg. hours of radium. The interval between treatment is one week. The dosage

of radium is estimated as being the minimum amount which will give beneficial results as determined by the experience of other investigators.

It was planned to give routinely six applications of radium. This is varied, however, and more applications are given if the case under consideration has been of long duration and the pathology is extensive.

The fact that each and every one of the 11 patients reported for the whole course of treatment is indicative of the fact that they must have obtained some benefit. Usually these patients, made miserable by the severe itching, shop from one hospital clinic to the other in the effort to obtain relief, but it is significant to note that all our patients reported regularly.

Observations indicate that patients required at least three or four applications before the desired relief from symptoms was manifest. There was no question in his mind that the patients all obtained marked symptomatic relief, which was gradual in character, but perceptibly evident by a gradual diminution of the discharge of lacrymation.

A recession of the pathological findings was not especially noted until all the radium applications had been given. Attempt was now being made to prove definitely whether radium will cure vernal conjunctivitis. Many investigators have reported cures. It must be remembered, however, that a large proportion of cases recover spontaneously without medication.

By way of argument, and realizing that vernal conjunctivitis is an ocular manifestation of an allergy, it is hard to conceive that the removal of the pathological state during one season of attack will prevent the occurrence of another attack the following season. In other words, it must be necessary for the radium to desensitize the patient against the susceptibility to the various proteins responsible for the disease.

Dr. Lehrfeld said the investigations would continue in the effort to prove or disprove the specificity of radium in vernal conjunctivitis. He felt, however, that radium has tremendous effects in alleviating the symptoms, principally that of itching.

Dr. Moore (in closing) said that he appreciated Dr. Shumway's point of properly gauging the dose of radium, as this is the most important thing in the treatment of these cases. If not properly gauged the lashes will drop out.

He also thanked Dr. Cross for bringing his case to be shown in contrast with the one he was presenting. In respect to Dr. Lehrfeld, as to using radium element in doctors' offices, he felt that it would be much wiser to refer cases to radiologists who are used to handling and gauging the proper dosage for these cases.

Ephedrine and Glaucoma

Dr. A. Barlow (by invitation) said that a possible causal relationship between ephedrine and glaucoma was suggested by the occurrence of acute glaucoma in 5 patients after ephedrine had been used in the nose for suspected sinusitis. The patients, 4 females and 1 male, were all past middle life, when the susceptibility to

glaucoma increases. It is possible, that while ephedrine may have no effect on intraocular tension in young persons and in persons with no glaucomatous tendency, it may act as an exciting factor in elderly people with a glaucomatous predisposition.

In view of the widespread use of ephedrine and the seriousness of the question involved, this clinical observation is reported to focus attention on the subject, and in the hope that other cases and experiments which will either confirm or negative this possible relationship will be reported.

MARCH 20

Differential Diagnosis Between Tumor and Choroidal Detachment

Dr. George Schwartzkopf, of Atlantic City (by invitation), presented a case. The patient, a man, aged fifty-two years, had been seen in the office on the previous day. There were vague complaints about visual disturbances in the right eye. The history revealed no trauma, important hereditary factors nor general disease.

The right eye looked normal, with no sign of inflammation. Iris and lens showed no change. In the peripheral lower part of the eyeground appeared a dark-brown bulging mass with definite limits up and inward, while temporal limits were indistinct. The prominence of this mass could be seen with + 13 to 15. Small, white, reflected areas with two medium-sized bloodvessels could be easily distinguished. There were no visible folds or tears. The eyeground was otherwise normal, with distinct arteriosclerotic vessels. Tension was normal and transillumination did not show any distinct shadow. There was a vision of 10/15 with a slight restriction in the field corresponding to the described area.

Discussion. Dr. Zentmayer said regarding the procedure of puncturing the mass for diagnostic purposes, that it brought to mind an experience he had had at the Wills Hospital some years ago, in a young man with doubtful serous detachment of the retina. Puncture was practised and the case proved to be a sarcoma. There was metastasis into the orbit soon after enucleation of the eye He felt that possibly, in puncturing the tumor, some

of the cells found their way into the orbit and thus caused the metastasis. Therefore, he considered this procedure not without some danger.

Dr. Langdon said one condition present, although he did not think it a factor in the general picture, was the advanced change in the retinal arteries. Whether that could have any influence on the condition he did not know, but thought that intraocular hemorrhage should be considered.

Tendon Transplantation for Paralysis of the Superior Rectus

Dr. Luther C. Peter reported a case of paralysis of the left superior rectus muscle improved by tendon transplantation, because he considered it somewhat unusual and because of several other features of interest.

Diplopia developed in July, 1928, and increased during the next month. Associated with the diplopia was a marked exophthalmos of the left eye—4 mm. of proptosis being present.

The patient's head is tilted to the right shoulder and the face is turned up and to the left. The diplopia field shows vertical diplopia of maximum degree up and to the left. In the upper right field there is slight crossing of the images, but there is little or none to the left.

The diagnosis manifestly is paralysis of the left superior rectus muscle.

Under local anesthesia the right superior rectus muscle was advanced, and the upper halves of the internal and external recti were split back for about 11 or 12 mm. and were fastened respectively to the internal and external borders of the superior rectus stump. The inferior rectus tendon was receded and reattached about 3 mm. posterior to the old tendon stump. The conjunctival wounds were closed by uninterrupted silk sutures.

Rotation fields before and after operation are of much interest. The patient can now rotate the left eye upward about 22 degrees above the horizontal, a gain of 40 degrees upward. The muscle balance shows 4 degrees of right hyperphoria and 2 degrees of esophoria at 6 meters, and 15 degrees of right hyperphoria and orthophoria in the lateral at 33 cm.

With proper head position the patient is now free from diplopia. In order to make him more comfortable, I propose to resect the right inferior rectus muscle.

The case is of interest, first, as to etiology, the paralysis probably

being associated with the proptosis of thyroidal disturbance. Second, the proptosis is characteristic of the persistence of exophthalmos after recovery from other symptoms of Graves' disease. It was a complicating factor during the operation. Third, the results are gratifying in a paralytic type of squint.

In 1921 I reported my first case of tendon transplantation before this Section. That was a case of paralysis of the external rectus of the left eye, in which the final rotations were 40 degrees internally and 50 degrees externally. Five other cases of external rectus paralysis, upon which I operated by similar methods since 1921, had varying results of from central fixation to 25 degrees of external rotation. The operation offers the only satisfactory correction in paralytic types of squint and is deserving of most careful study to improve the technic.

In the 7 cases which I have operated on by a modification of the O'Connor technic, the variations which seem to have given me the best results are, first, the transplanting of one-half of the tendon instead of a smaller strand. In my earlier cases, I transplanted too small a part of the tendon. A generous half seems to offer better prospects of good rotation in the field of the paralytic muscle. The other variations are, second, a very free advancement of the paralytic muscle, and third, complete tenotomy or recession of the antagonist. In paralysis of the external rectus, it is possible to completely tenotomize the internal rectus without danger of creating a paralytic divergent squint. In paralysis of a vertically-acting muscle the tenotomized tendon should be securely attached to the globe.

Discussion. Dr. Chance spoke of the case of congenital paralysis of the superior rectus in a young girl who was exhibited at the Section about eight years ago. He did just such a transplantation as Dr. Peter recommends, but he took much more from the lateral muscle than what had been directed in the descriptions of technic to be employed. The patient was seen two years ago; she had been free from any deformity or diplopia. She also had had a sort of compensatory ptosis, which was relieved by the operation on the ocular muscles. As she had never been able to see above the horizontal line of vision with the left eye, the operation had made it possible for her to raise the eyelid. Altogether, the procedure is a most satisfactory one.

Dr. Shannon congratulated Dr. Peter on the splendid result obtained in his case of paralysis of the superior rectus. In this connection, Dr. Shannon reported a case of complete paralysis of the external rectus on the right side in a patient forty-nine years of age, who had suffered a fracture of the

skull eight years previously. The paralysis followed the injury within a few days. The eyeball was deviated inward 50 degrees, with no power of outward rotation. The superior, external and inferior recti were exposed, and a tuck made of the external rectus. The superior and inferior recti were then split and the temporal slips sutured to the tendon attachment of the externus. The internus was tenotomized. A week following the operation the eyeball could be rotated outward to the midline and slightly beyond. Binocular single vision was obtained in certain positions and the eyes appeared approximately parallel.

Dr. Peter (in closing) said that Dr. Shannon was very modest in his report. The result in his case was really very good, as previous to the operation the patient had been unable to rotate the eye at all, even to the front. The diplopia is now relieved. Dr. Peter believed that had a complete tenotomy of the internal rectus been practised, the patient might have considerably more external rotation. He was also of the belief that an advancement of the external rectus, instead of a tucking, would have added much to the case.

Foreign Body in the Lens

Dr. George Cross exhibited the case of foreign body in the lens.

Discussion. Dr. Zentmayer said that in the fall of 1927 Dr. Fordyce brought to the Wills Hospital a man whose eye had been struck by a piece of steel a few hours previously. There was a perforating wound of the cornea, a wound in the capsule of the lens and a foreign body situated within the lens, almost at the posterior capsule. There was no opaque track in the lens. The foreign body was withdrawn by a magnet and for several days afterward there was no opacity in the lens, but later the lens had to be removed on account of secondary glaucoma.

Dr. Heed said he had seen foreign bodies retained in the lens for some time without any general opacity, and felt that this was one place in the eye that may be treated conservatively so long as there is no loss of vision. He exhibited a small foreign body which had produced a reaction thirty hours after being imbedded in the lens. Vision reduced to 20/100. A definite star-shaped opacity and the foreign body clearly seen posterior to the subcapsular area, up and in. There was a fair view of the fundus, but with the rapid change in the lens and the loss of vision it was felt that the sooner the steel was removed the better, and operation was performed thirty-six hours after the injury.

Variations of the Intraneural Course of the Central Vein of the Retina

Dr. W. E. Fry (by invitation) presented his observations made from the examination of serial sections of 36 optic nerves, obtained from 20 cases. The sections were cut 20 microns in thickness and

in some instances every fifth, and in other instances every tenth section was prepared and examined. Horizontal sections were made, beginning about 2 mm. behind the globe and extending nearly to the intracranial portions of the nerve.

The artery and vein pass straight posteriorly through the center of the optic nerve from the level of the lamina cribrosa, usually, but not always, close together. At a variable distance from the lamina the vessels turn and proceed toward the periphery of the nerve. Based upon the course at this point, two types may be distinguished. We may speak of type one, in which both the artery and vein proceed to the same point on the arc of the periphery of the nerve; and of type two, where they proceed to different points on the arc of the nerve. In this second type the artery and vein diverge from each other, usually at an angle of 90 degrees, although it may be slightly less or greater than this in some instances. The artery and the vein proceed to the periphery as indicated by the first type in 15 nerves, or 42 per cent of the instances, and as in the second type in 21 nerves, or 58 per cent of the instances.

In the first type, when the vessels reach the point of turning, they make a right-angle turn with relation to the length of the nerve stem. Both vessels usually crossed the intervaginal space and pierced the dura immediately upon reaching the periphery of the nerve.

In the second type the vein approached the periphery of the nerve anteriorly to the artery in 81 per cent of the instances. The artery crossed the intervaginal space immediately upon appearing on the periphery of the nerve, and in all instances crossed the space before the vein, even though the latter had appeared in a subpial position while the artery was still within the center of the nerve.

In 4 of the cases of the second type, the vein, after coursing posteriorly in the pia, turned at right angles about the periphery of the nerve, passing through an arc of a quarter of a circle and made its exit from the dura at a point corresponding to the line of exit of the artery.

Discussion. Dr. T. B. Holloway stated that from the standpoint of orbital anatomy he thought this contribution was of the most importance. It also had distinct bearing on the clinical side. As Dr. Fry intimated, it opens up the possibility that more of our cases of thrombosis of the central vein may have their focus further posterior, as has been suggested by Yamaguchi.

Aside from the clinical bearing on brain tumor cases, the findings immediately suggest the cause of the retinal hemorrhages in cases of hemorrhage into the sheath. Uhthoff and others have contended that it is impossible for the blood to penetrate from the sheath, while Paton believes it may follow along the lymphatics at the lamina cribrosa. While this latter statement may be true, these findings seem to impress one with the probability of a retinal vessel origin in a definite number of cases.

APRIL 17

Case of Acute Vitreous Haze Following the External Use of Adrenalin and Scopolamin for the Separation of Synechiæ

Dr. H. Maxwell Langdon presented the following case report: Mrs. E. T. L., aged seventy years, was first seen on July 10, 1920, with a low-grade iridocyclitis of the left eye. The right eye was quiet. Under local treatment the attack was soon overcome. The attack was thought to be due to ethmoidal infection. The sinus condition was treated by suction.

On September 20 there was a similar attack in the right eye. There were recurrent attacks during the rest of 1920 and through July, 1921. At this time the left ethmoid was operated upon and she was free from attacks until July, 1924. Following an attack in July, 1924, the left sphenoid was opened with good results.

An attack occurred in the right eye in 1927 and again in 1929. In March, 1930, a severe attack occurred in the right eye. The use of heat and scopolamine for forty-eight hours having no influence on the pupillary condition, a pledget of cotton moistened with a few drops of a 1 to 1000 adrenalin and 2 drops of 0.5 per cent solution of scopolamine were placed beneath the upper lid.

Before this there had been some haze in the anterior part of the vitreous. The vision, however was 5/5 plus, when corrected. In fifteen minutes the pupil was widely dilated, but the upper part of the cornea was hazy, the vitreous opacities had greatly increased and vision was reduced to 5/45. The cornea cleared in half an hour and in twenty-four hours the vitreous was nearly as clear as before this dilatation. Vision was 5/6. The right ethmoid was cleared and the vision was 5/5.

Discussion. Dr. Zentmayer said that he had frequently used the method spoken of by Dr. Langdon and had used adrenalin repeatedly in dilating the pupils for ophthalmic purposes and also for the treatment of glaucoma. He had never seen the phenomenon Dr. Langdon has spoken of. He also said that recently a paper had been read before the Section calling attention to cases of acute glaucoma following the use of ephedrin. He had never seen acute glaucoma following an extended use of this drug.

Dr. deSchweinitz said that, although he had not observed the phenomenon which Dr. Langdon describes, it was not inappropriate to refer to a corneal condition not infrequently noted when adrenalin and a mydriatic (atropin or scopolamin) are employed to break synechial attachments, the adrenalin being applied by means of a small roll of cotton placed beneath the upper lid (Gradle's method), namely, a delicate but definitely slightly smoky corneal haze.

The first time he had noticed this haze (a patient with relapsing iridocyclitis) he thought it indicated that a rise of tension was taking place; in short, that the haze was like the one observed in the prodromal stages of acute glaucoma. But tested, there was no rise of tension; the slitlamp showed a delicate edema of the corneal epithelium. The haze rapidly disappeared.

These phenomena had been noted by Dr. Baer and himself on several occasions in his office, and Dr. Baer had made similar observations in Wills Hospital. The haze might be explained by a temporary alteration of the intraocular tension or by a direct effect of the drug on the corneal epithelium.

Some Interesting Cases of Sclerocorneal Trephining in Chronic Simple Glaucoma. (A Clinical Paper)

Dr. William Zentmayer: The following cases of chronic simple glaucoma in which sclerocorneal trephining was done are considered of interest clinically as illustrating:

(*a*) The frequently satisfactory results of this operation.

(*b*) Quiet iritis. A complication which does not always defeat the purpose of the operation, but which frequently interferes with a satisfactory visual result.

(*c*) Transient marked changes in refraction, an annoying consequence of this operation.

(*d*) The loss of the eye from late infection, the dreaded sequel of the operation.

Case I.—*Chronic Simple Glaucoma.* Bilateral trephining with preservation of practically full-form field and no enlargement of the blind spot in one eye, over a period of ten and a half years; and moderate contraction with marked enlargement of the blind

spot in the field of the other eye; marked variations in the refraction of one eye; and the disappearance of the glaucomatous cup in one eye.

CASE II.—*Chronic Simple Glaucoma.* Sclerocorneal trephining of the right eye. Iridectomy in the left eye. Marked variations and increase in static refraction of the trephined eye.

CASE III.—*Chronic Simple Glaucoma.* Sclerocorneal trephining of the left eye. Immediate and progressive loss of vision from a low-grade iridocyclitis, resulting in marked deposition of pigment over the tissues of the anterior chamber.

CASE IV.—*Subsiding acute glaucoma* in the right eye. Unsuccessful sclerocorneal trephining. Successful iridectomy.

Chronic simple glaucoma in the left eye. Sclerocorneal trephining with pigment migration over the tissues of the anterior chamber and development of subcapsular opacities.

CASE V.—*Chronic Simple Glaucoma.* Bilateral sclerocorneal trephining. Loss of right eye by late infection, and of the vision of the left eye through progress of the disease in six years' time.

CASE VI.—*Chronic simple glaucoma* in both eyes. Sclerocorneal trephining of the right eye. Early rapid increase in static refraction with failing vision, and later acute secondary glaucoma from iridocyclitis.

DISCUSSION. Dr. deSchweinitz said that it was his experience that "quiet iritis," after corneoscleral trephining, was less apt to occur if part of the operative technic had been a complete iridectomy; but it did occur in a certain number of cases, in greater or less degree, even if a mydriatic had been instilled at the very beginning of the postoperative treatment. Another complication was the development of lenticular opacities, even after technically correct operative procedures; but should a complete cataract supervene, it was usually possible to extract it with success. He quoted cases of this character in his and his associate, Dr. Baer's, experience.

Dr. Cowan said that the changes in refraction to which Dr. Zentmayer referred could hardly be due to change in the position of the crystalline lens.

Based on the values for Gullstrand's schematic eye, the crystalline lens displaced 1 mm. forward would increase the dioptic power of the eye 0.65 D.; 2 mm. displacement would make an increase of 1.30 D. If the cornea were only 0.5 mm. in thickness, so that with the lens displaced 3 mm. forward, leaving the anterior chamber only 0.1 mm. in depth, the increase in refraction would be only 1.88 D., correctable by a concave lens of less than 2 D.

Dr. Holloway said, regarding Dr. deSchweinitz's reference to quiet iritis in these cases, that it has been his experience in a majority of cases; and while he had not been as much impressed with its frequency following

complete iriedectomy, he has found that if you examine the lower portion of the iris you will find that it is present.

He said that he had hoped Dr. Zentmayer would cite some cases in regard to the filling-in or sudden plugging of the trephine opening, such as not infrequently occurs, sometimes due to proliferation of the uveal tissue or possibly blocking with a ciliary process. Only recently he had had an unfortunate experience in a patient who had been under observation for many years, coming to him at the age of twenty-nine years for glaucoma. Bilateral iridectomy was done with good result. She was carried along for eight or nine years, when a cyclodialysis was done in each eye and the tension reduced below normal, subsequently rising rather slowly over a period of three or four years, until recently it rose to 60. Under these circumstances trephining was done over the outer pillar, tension remained down for two weeks and she was about to be discharged when, overnight, the tension rose and remained up for five or six days and she was again trephined over the outer pillar of the coloboma. It is too early to say what the outcome will be.

He said, also, that he had never had a secondary infection as a complication of trephining and he felt that it was very much overrated and not particularly apt to happen; at least this had been his experience, and he felt that if a check-up of these cases were made it would be found to be a rare occurrence.

Dr. Langdon said, concerning extraction of the cataract which comes on after trephine or which is increased by trephining, that he had had two experiences in removing them—one successful, with very satisfactory vision; the other, a perfectly satisfactory section was made, but attempt to extract the lens was blocked by the size of the lens. Pressure on the cornea produced change in its circulation, with degenerative keratitis. This lens was the largest that he had ever seen and it seemed probable that the size of the lens had something to do with the production of the increased tension, and possibly in such cases a lens larger than normal might be expected.

The Treatment of Argyrosis. Exhibition of Case

Dr. I. S. Tassman (by invitation): A patient from the clinic of Dr. J. M. Griscom, at the Wills Hospital, was treated for argyrosis of the conjunctiva according to the description of a method by W. F. Weymann, at the meeting of the Ophthalmological Section of the American Medical Association in 1929. Mrs. L. S., an Italian woman, had a rather severe argyrosis of the bulbar, palpebral and tarsal conjunctiva of both eyes, but more marked in the right. She has to date received two injections, each 0.6 cc. of a mixture of 2 parts of potassium ferricyanide, 2 per cent, and 1 part sodium thiosulphate, 12 per cent, as described by Weymann. There resulted

in the right eye a noticeable clearing of the discoloration of the conjunctiva injected. A comparison could be made with the left eye, which was formerly the better and now the worse of the two. The presence of the argyrosis in this patient was due to repeated treatment of the eyelids with nitrate of silver for a period of months a number of years ago. The patient is to receive subsequent injections of the mixture described. There was little reaction and pain following the injections, and the procedure can be carried out practically without incapacitating the patient.

Ophthalmic Operations

Dr. Frank C. Parker (by invitation) showed some very interesting moving pictures of ophthalmic operations.

Discussion. Dr. Holloway said that as he had had some experience with moving-picture camera work in relation to medical subjects, he knew just what Dr. Parker's accomplishment meant to him and how difficult pictures of this nature are to obtain, and that he wished to congratulate Dr. Parker on his success.

He agreed with Dr. Parker that the moving picture has come to stay, and that as yet its place in relation to experimental medicine, bacteriology, etc., has not been attained, and that he felt much was yet to be accomplished.

Dr. Chance said he wished to congratulate Dr. Parker on his success, and he felt that much was yet to be accomplished through the perfection of the moving-picture machine in relation to the teaching of medicine.

Dr. Parker's machines are in the crude stage of the early models, yet so accurate are they in their adjustments that it is doubtful that the elegant and highly-polished products about to be put on the market can be capable of greater satisfaction than is shown by the one exhibited tonight. In this connection, Dr. Chance was reminded of his association with an aged English oculist, at Amsterdam, who intended to visit Utrecht, to go once again to Donders' laboratory to see the simple and quite crude appliances which Donders had employed while Dr. Chance was his student many years ago—the same instruments with which that great physiologist had so accurately and precisely obtained his results.

Congenital Total Color-blindness

Dr. W. E. Fry (by invitation) said that congenital total color-blindness is a moderately rare condition. Up to the present time there have been but four reports, which include 7 cases of this condition from this country.

These cases are unusually uniform in the characteristics which they present. The following six points are the ones most frequently noted: Amblyopia, photophobia, nystagmus, shortening of the red end of the spectrum, displacement of the brightest part of the spectrum from the yellow toward the green, and frequent central scotoma.

Case Report.—Miss C. T., aged twenty-three years, was first seen in Dr. Holloway's office August 28, 1929. She was later studied at the University Hospital. She stated that on a bright day her vision was so poor that she was unable to go about alone, but that in dim light in the evening she was able to get about very well. There was apparently no necessity for dark adaptation. She was totally color-blind. To her, different colors had a different amount of brightness, but she was unable to name colors from their different luminosity. Her vision was 6/60 in the right eye and 6/30 in the left. Her eyes were negative externally and there were no fundus changes. The visual fields for one and two degree white were normal in size and outline and there was no central scotoma. The luminosity curve corresponded to that of the achromatic scotopic luminosity curve of a person with normal color-vision.

Discussion. Dr. deSchweinitz said that total congenital color-blindness, the eyes being normal in all other respects, must be very unusual. He had seen one such case, about thirty years ago—a healthy woman of Quaker extraction; eyes normal in all respects, with practically complete color-blindness. Because of the deep interest of the late Dr. William Thomson in all matters pertaining to color vision, the patient was referred to him for study. What the ultimate results were he could not report, as she did not return either to Dr. Thomson's or his care.

It would seem that such patients must be excluded from the congenitally totally color-blind, who have diminished visual acuteness, often dread of light, nystagmus, and in whose visual field scotomas can be demonstrated, but, curiously enough, dark adaptation remains normal.

Dr. Chance said he wished that some other title for this clinical entity might be had; "color-blindness" has a pretty well-fixed clinical aspect. In ordinary color-blindness, as a rule, there are no pathological changes; the patients come because they are simply unable to distinguish colors. He said that he had found it difficult to make a complete study in private cases, as such patients are rather sensitive about their condition and reject, if they do not actually resent, overtures to submit to the tests. He cited instances in his experience occurring in those who were strict adherents to certain pietistic religious customs, among whom numerous well-known cases have arisen, evidently dependent upon blood relationship from the intermarriage of adherents.

OCTOBER 16

Résumé of the Special Inquiry of Eye Health in Philadelphia

Dr. Haven Emerson, of New York City, after referring to the high standing of Philadelphia as an ophthalmological and medical center, recalled the fact that Philadelphia led in making an extended study of the eyesight of school children as far back as 1878, under the chairmanship of Dr. Samuel D. Risley, and that a survey of lighting conditions in public schools had been conducted actively in 1875. Quoting from an editorial in the Philadelphia *Press*, under date of September 20, 1878, Dr. Emerson pointed out how shrewdly Dr. Risley's survey committee secured the coöperation of the press in preparing the minds of the public for this first study of the vision of school children. The work of Dr. Risley's committee was monumental and served as a guide all over America.

Dr. Emerson then reviewed the general hospital and health survey work of the city, pointing out how nearly every phase of that study had some lesson in it of value in eye health. He featured particularly the social-hygiene survey and the notorious abuses of quacks and charlatans in taking advantage of the gullible, pointing out that Philadelphia is really a center for retail distribution of misinformation conducted on a scale not found exceeded anywhere in America.

Subsequently, Dr. Emerson discussed in considerable detail the lack of complete reports by physicians, hospitals and others of the occurrence of sore eyes in babies. He complimented particularly the work of the Visiting Nurse Society in this as in all other phases of their work, and concluded his discussion with a strong plea that the Section of the College should maintain a standing committee to coöperate with the Health League in securing adequate support for the city health department in promoting a much needed extension of its eye health services and in improving conditions so obviously needing correction in the public and parochial school systems.

Discussion. Dr. B. Franklin Royer followed Dr. Haven Emerson and discussed in some detail the incompleteness of the dispensary records in practically every eye clinic where histories were reviewed. He stated that

with the aid of the ophthalmologists on the committee a sort of measuring stick was designed by using thirteen different conditions which all were agreed would require watchful care in the eye clinic and many of which would be likely to cause deterioration of vision if neglected. The ophthalmologists of the committee estimated the number of return visits that ought to be expected in each condition and the duration of treatment; then, with the aid of a well-trained statistician and the complete coöperation of various persons charged with the responsibility for these records in the outpatient departments, some 738 records were reviewed and contrasted with the norm established by the committee. It was distressing to find such a tremendous waste of professional effort shown throughout all of these clinics. So large a proportion of the patients attended a totally inadequate length of time that the physician was given no chance to demonstrate what could be done by curative medicine, and a large proportion of the patients were lost to the clinic without showing what result, if any, had been secured. Many patients were found with serious eye conditions, such as interstitial keratitis, keratoconjunctivitis, phlyctenular choroidal disease or uveitis, with attendance continuing scarcely long enough to establish the diagnosis.

Out of the 378 cases reviewed only 4 were recorded as having made the desirable number of dispensary visits: 1 of these was a case of tuberculous keratitis, 1 a case of iritis, 1 of disease of the choroid and retina and 1 of glaucoma; 40 cases of corneal ulcer (ordinary abrasions not being included), 16 cases of interstitial keratitis, 33 cases of keratoconjunctivitis phlyctenular, 21 cases of iritis, 7 cases of uveitis, 54 cases of diseases of the choroid and retina, 6 cases of papillitis, 2 cases of papilloedema, 26 cases of optic atrophy and 12 cases of glaucoma made but a single clinic visit.

The point brought out by the speaker was that the efforts of ophthalmologists are largely wasted unless a trained medical social worker serves as the go-between to interpret to the family the scientific lesson which the doctor wishes to impress, but in language the patient may readily understand, and to see that those family adjustments are made which will permit return appointments to be kept until such time as the doctor has had an opportunity to treat the condition adequately.

The lighting conditions in the public and parochial schools and the shade adjustments were discussed in considerable detail, emphasis being placed upon the fact that Philadelphia was found to be tolerating lighting conditions in some of the public schools today that were severely condemned by the survey group in 1875. It was pointed out that in some of the newer school buildings there was need of eliminating windows in the front of the room which were the source of annoying glare; that window shades at the time of the survey were almost universally of the single-roll type hung from the top of the window, instead of double-hung at the middle, one rolling up and the other down; and that the old-fashioned method of arranging desks with the left side parallel with the windows existed, with most of the desks fastened to the floor in this manner, while modern school practices would indicate that the desks should be so oriented that the pupil faces a little toward the inner front corner of the room rather

than toward the straight front wall, in order to bring light properly over the left shoulder and prevent glare in the eyes of those seated in the rear rows near the windows.

On behalf of the survey committee doing the work, as well as of the National Society for the Prevention of Blindness, Dr. Royer expressed great appreciation to hospitals, dispensaries and other agencies in the city for their splendid coöperation. Everybody helped.

Mrs. Winifred Hathaway reviewed the survey of the rooms in which sight-saving classes are conducted and of the equipment found in use, and pointed out that, through economies in utilizing buildings not available for other purposes, some gross faults had been tolerated. She featured the fact, however, that, following the survey, nearly all of the serious faults had been speedily corrected. She pointed out the need for ophthalmologists to keep in closer touch with the activities, customs and practices of the sight-saving class, both for the purpose of encouraging and guiding the teacher and in order that the practising oculist might know more about the advantages of the sight-saving class.

Dr. L. C. Wessels (by invitation) gave a résumé of the work done in the Eye Clinic at City Hall.

NOVEMBER 20

Emergency Outfit for Ophthalmic Examination and Treatment

Dr. Edward A. Shumway showed an emergency outfit for ophthalmic examinations and treatments away from the office or hospital. A stock physician's bag had been fitted with a rack holding six small bottles for ophthalmic solutions, and had additional provisions for holding a cotton container, solutions of boric acid, etc., and would hold dressings and instruments. The outfit could be obtained through Wall & Ochs and had proved very practical.

Trachoma in Wrestlers

Dr. Zentmayer reported a case of recent trachomatous infection of the right eye of a wrestler. The patient had been an athletic coach and had only recently gone in for professional wrestling. He was aware of the prevalence of trachoma among wrestlers and was not surprised when this diagnosis was made of his trouble.

Dr. Zentmayer called attention to the contribution of the late Dr. Patton, eight years ago, who saw, with the late Dr. Gifford, 8 cases of trachoma in wrestlers. It was Dr. Patton's belief that

the mode of transmission was that in the course of wrestling matches the contestants perspired freely and, when the referee was not looking too closely, often massaged the opponent's eyes, as Dr. Patton suggests, to produce unconsciously the oculocardiac reflex.

It seemed proper to make this report so as to again call to the attention of ophthalmologists this mode of transmission of trachoma that they may bring it to the notice of the health authorities of their respective communities and thus endeavor to eliminate this source of infection.

Discussion. Dr. Mann said that during the early part of last summer he had occasion to observe 2 cases of wrestlers who had been wrestling on various occasions. The first case was one who stated that his eyes had never been inflamed until he was being examined for a bout in one of the southern states. He was the last among four or five examined. The examiner evidently did not remove his gloves. The man noticed the fact and told Dr. Mann about it. Those other wrestlers, no doubt, had some acute inflammatory condition, whether trachoma or not he did not know, but two or three days later he developed an acute conjunctivitis, and when he came to Philadelphia he was seen by Dr. Mann. The case did not turn out to be trachoma, but was an unusually acute conjunctivitis.

The second case was one of much longer duration, although the wrestler had been in the profession for a number of years and had tracheoma for some years. The appearance of the conjunctiva in his case did not show the follicles at the time he was examined. On everting the lids the conjunctiva rolled out in thick red folds in the upper and lower lids, particularly the lower.

This man said that trachoma was quite prevalent among all the wrestlers and mentioned the names of some who had trachoma; yet they are examined before a bout. Of course, some of the cases are not trachoma; where the head is being banged against the floor the dust arising enters the eyes and causes chronic conjunctivitis, which is a part of trachoma. But many of the cases do have trachoma, and this second one had, and it was stated that many are being treated constantly wherever they go.

Dr. M. E. Smukler said that during the past two years he had seen a case of trachoma in a wrestler who at that time was the champion of the world; one eye was affected. The case was reported to the Bureau of Health. After the case was reported this man left the city for about six months. During his absence his lid was rolled and treated. He was discharged by his oculist as cured.

The question came up, by the Department of Health, as to whether he should be allowed to continue his profession in this city. Examination by the Bureau of Health revealed that there was no active inflammation present and no discharge from his affected eye, so they did not deem it advisable to bar him from continuing his profession with the present condition of his eyelids.

Dr. Maxwell Herman commended Dr. Zentmayer for bringing to their attention a condition which seemed to exist and apparently seemed to be common knowledge among professional wrestlers and their training establishments and which they themselves have designated as "wrestlers' sore eyes."

He said that in the past two years he had had as patients professional wrestlers, of whom undoubtedly 2 had trachoma. One was a case similar to Dr. Zentmayer's—a man of some education who, in his college days, had been an athlete of quite some intercollegiate standing and who had entered professional wrestling as a means of livelihood. He stated that he became infected with trachoma in the training establishments and that he recognized the needlessness of such infection, but since the culprits in this matter have been foreigners who have brought in the infection, but who were champions and whose power and popularity were such that the little fellows could offer no protest. Dr. Herman said there was little doubt in his mind that some of this so-called "wrestlers' conjunctivitis" is true trachoma and Dr. Zentmayer's paper and the publicity of this question in the profession may bring forth a great deal of good and justice to the professional wrestler.

Incorporation in the Prescription of the Style and Shape of the Lenses

Dr. Alfred Cowan read a paper in which he urged the ophthalmologist to incorporate in his prescription the style and shape as well as the effective power of the lenses he desires. [The paper will be published in full.]

Discussion. Dr. Sidney L. Olsho said that in convex spherical meniscus lenses up to +8 the −6 inside curve is constant and the spherical lenses are so supplied without any trouble by all optical shops. Also, convex plane cylinder toric lenses have a constant −6 inside curve. But when a convex toric spherocylinder is to be ground, the front surface remains +6 combined with the cylinder, and the spherical effect is obtained by diminishing the minus of the inside surface instead of increasing the convexity of the front surface.

The objections to this method have been stated. Why is it persisted in? It is not common knowledge that a constant −6 inside curve for powers up to +8 would provide a more efficient and wider angle lens?

The answer is that it is easier. When toric spherocylinder lenses superseded the flat forms they came almost universally, for commercial reasons, to be ground with a constant +6 front-surface base curve. Throughout the country the optical shops now have this set-up; they have the tools, they have the lens blanks and they have the habit.

To change to a constant −6 inside-base curve for convex spherocylinder toric lenses up to +8, as suggested by the speaker, is not a simple matter, but means revolutionary changes—the purchase of hundreds of new blanks

and tools; an unanimous discarding of the somewhat faulty but rapid method in vogue.

Instead of taking a single step forward, the national manufacturers have gone many steps farther by making point-focal lenses universally available at only a slight advance over the present prices of ordinary toric lenses. This has been brought about by production on a huge scale and standardization.

True it is, that some of the claims made for these lenses have been exaggerated, but it cannot be denied that they are a great advantage, particularly in the higher foci. They do afford a wider angle of efficiency than lenses less well balanced. Furthermore, the polish of these lenses is much finer.

This advance, all along the line, in lens designing and lens marketing would be delayed for many years were the commercial incentive to the manufacturers not present. The low price of the toric lenses we now use is dependent upon the mass production of lens blanks and molds by the same national manufacturers. In the bifocal field we are using proprietary types without giving the matter a thought. The several concerns marketing point-focal lenses employed distinguished scientists to work out the best possible curve combinations. They followed in general the teachings of Tscherning. The results are not ideally perfect; there are commercial and technical limitations.

Let me turn back again for a few moments to the discussion of toric lenses. Some of the larger optical shops have the tools not only to grind with a +6 base-front curve, but also with +3 and +9 base curves. This increases the range in which these opticians can operate in an attempt to equalize inside curves of an unusual lens pair. It also affords at times a means of approaching the most efficient relation of inside and outside curve. Then by transposing, as has been pointed out by the speaker, we may often secure more efficient combinations. He said he wished to point out that these surfacing specifications often involve special and costly grinding. A real point-focal lens would in many instances be less costly and perhaps better.

In the several point-focal series of lenses the surfacing problems have been worked out more or less in advance. This helps those not familiar with the juggling of base curves and transpositions. The point-focal lens may not be ideal, but the error will be fractional. I doubt whether a specially surfaced ordinary toric lens would be more perfect. If a lens requiring unusual surfacing must be replaced in any but the original shop, the chance of not receiving the identical surfacing is very high. A standardized lens is more apt to be replaced without error.

Dr. George S. Crampton said that, as usual, Dr. Cowan has covered the subject most thoroughly, and he felt sure that all were looking forward to a chance to peruse his paper when they had time to go over it carefully. He said that here in Philadelphia we are particularly blessed with guardian angels in the way of most wonderful opticians, and that he did not believe there was another city in the country in which the oculists are so well guarded by opticians who do beautiful work. He understood that in

Chicago there is only one retail concern that does not refract, while here there are hordes of them.

Dr. Crampton said that Dr. Cowan's paper covered the subject thoroughly, and the question of going into further figures is quite uncalled for, but that it makes us stop and think, however, and quite rightly, that oculists should not depend on the opticians. Their cases may go to opticians who will take care of these transactions to the best advantage of the patients, but this is not always so. We know that frequently our prescriptions get into the hands of inferior opticians, and they get the sort of compounds which are almost "poisonous," as Dr. Cowan pointed out.

He felt that this paper was very timely, and that it was for them to think more seriously of this matter and not be in the position of a man to whom he had been talking, who when asked how do you prescribe your lenses, answered:

"All toric."

What about the spheres?

"Toric also, everything toric."

Dr. Cowan's paper also leads one to think of possible slips in the writing of actual prescriptions. As a rule, many make their prescriptions a little mysterious. A simple form is best. Dr. Crampton said that you may write combination marks and two or three lines for sphere. In the end, when the optician gets it, he will write a very simple prescription. The simplest form would be "+50 +1.00 cx 90." Some write the sphere above and the cylinder below, so there will be no mistake. Some oculists are in the habit of not putting names on prescriptions at all.

Dr. Cowan spoke of 3 and 9 base curves as being more expensive; about one dollar a lens more. Some specify curves, or go into great detail and show that they know the whole optical side of the question. Others are less careful. Some give the size of the bifocal segment and others leave it to the optician. Some give the center for both the distance lens and the bifocal segment. As far as writing the prisms is concerned, that is pretty uniform, as they usually use the little triangle and afterward give the base. Some have a printed blank. As you know, the English oculists, and a good many in this country, depend on the little circle showing the axis. He said that he thought that started when foreign opticians did not know our system and it was better to mark the axis on prescriptions.

Dr. Crampton said this was really a plea for simplicity in prescription writing. If you have a lot of records to go over and want to find a prescription, it is better not to have the combination sign. They combine automatically and it just gets in the way.

Modified Le Grange Operation for Glaucoma

Dr. J. Milton Griscom presented the details of a modified Le Grange operation which he first used in November, 1929. The steps of this operation are as follows:

A broad conjunctival flap, including the episcleral tissues, beginning 8 to 10 mm. from the upper limbus, is dissected down to the corneal margin. While the flap is turned down over the cornea by an assistant, the point of a keratome is entered in the sclera 2.5 mm. behind the scleral margin of the cornea. As soon as the point penetrates the sclera the conjunctival flap is turned upward; the keratome is pushed forward on a plane with the posterior surface of the cornea until the assistant, looking from below, sees the point emerge in the anterior chamber. With the anterior surface of the iris as a guide, the keratome is then further introduced into the anterior chamber until the incision in the sclera is about 5 mm. wide, following which the instrument is quickly withdrawn. The anterior lip of the scleral section is then seized with an iris forceps and an elliptical piece of sclera, 1 mm. wide and 3 mm. long, excised with a pair of small curved scissors. This is followed by a broad complete basal iridectomy. The conjunctival flap is replaced and sutured.

Discussion. Dr. B. F. Baer stated that he did this identical operation first in December, 1929. Owing to the extreme shallowness of the anterior chamber encountered in many glaucomatous patients, the procedure required modification because of the inability to use the keratome safely. In those eyes with extremely shallow chambers an incision was made with a Graefe knife and the sclera slightly bevelled just before the completion of the section.

During the past eleven months some 28 to 30 eyes have been operated upon by one or other of these methods, with a subsequent rise in tension in 2 eyes. The tension has been easily controlled by again resorting to miotics.

In none of the cases has quiet iritis been observed, nor was there further degradation in central vision or the peripheral fields. The operation has been extremely satisfactory and far less dangerous than Le Grange's operation. In no case has prolapse of the vitreous or ciliary body been encountered.

Dr. deSchweinitz expressed his satisfaction in that Dr. Griscom had introduced to the Section his method of performing scleretoiridectomy for the relief of glaucoma, being an operation which is of definite value.

Shortly after David Priestley Smith, seventeen years ago, had published his technic, to which Dr. Baer had referred, Dr. deSchweinitz stated he had adopted a procedure much less complicated, being, in fact, one which was precisely similar to that which Dr. Griscom had described, and in all instances (always cases of chronic glaucoma) the results had been eminently satisfactory, although the operations had been few in number.

About a year ago he had suggested to his associate, Dr. Baer, a return to this method of operating, and the results, as the Section has heard,

were entirely satisfactory. In no case was there a failure to secure an adequate fistulous area, although in 2 cases there had been a return of increased tension, quickly controlled by miotics; quiet iritis had not developed in any of the operated eyes and in none of them had there been a dangerous lowering of intraocular tension.

It was interesting, he said, that two surgeons operating in the same hospital had been unconscious of the fact that they were utilizing an exactly similar technic.

Naturally, considerable additional experience with this modified Le Grange procedure would be necessary before its permanent value could be established.

Dr. Shannon said that the modified Le Grange operation, as suggested by Dr. Griscom, appealed strongly to him by reason of its simplicity. The classic Le Grange operation always presents the danger of cutting into the ciliary body in completing the section. This modified procedure, however, obviates to a great extent this danger. Some operators make the scleral section with the keratome before dissecting down the conjunctival flap. This, to his mind, accentuated the risk of introducing the knife beneath the iris, an accident that recently happened to a skilful operator, much to his chagrin. He further stated that he would be inclined to try out the operation at the earliest opportunity.

Dr. George H. Cross said he had never tried the modification of the Le Grange operation, as suggested by Dr. Griscom, and the results in the last 15 cases were satisfactory. These were all operated upon with the Ridley cataract knife, the sharp point permitting one to enter a very narrow anterior chamber and make the scleral section.

The Bacteriologic and Immunologic Aspects of Iritis, With Special Reference to Its Relationship to Bacterial Allergy.

Dr. John A. Kolmer read a paper on this subject, and attention was drawn to the lack of acceptable data bearing upon the etiology of chronic recurrent iritis associated with focal infection, and a plea was made for more systematized bacteriologic investigation in this disease.

1. Iritis of exogenous origin, as well as tuberculous and syphilitic iritis, may be regarded as well established from the etiologic standpoint, but the iritis of focal infection still requires a great deal of investigation for ascertaining the exact mechanism of infection.

2. The opinion was expressed that chronic recurrent iritis was probably a streptococcus infection in the majority of instances, with some cases due to the gonococcus, staphylococcus, pneumococcus and other organisms.

3. The occurrence of streptococcus iritis secondary to primary streptococcus infection in the tonsils, apices of teeth, etc., does not necessarily involve acquired selective affinity of the organisms for the iritis, although the experimental production of iritis among rabbits, by intravenous injections of broth cultures of streptococci from such primary foci, strongly suggests this possibility.

4. It is probable that toxins elaborated in primary foci may be responsible for iritis, rather than the bacteria themselves, and an experimental method for studying this phase of the problem was outlined.

5. Attention was drawn to the points of resemblance between iritis and acute and chronic arthritis of streptococcus origin, with the possibility of iritis being due to acquired allergic sensitization of the iris to streptococcus toxins.

6. It was suggested that autogenous vaccine therapy be employed routinely in the treatment of chronic recurrent iritis when streptococci, staphylococci or pneumococci are recovered in cultures of probable primary foci, as removal or drainage of primary foci alone may not result in recovery.

7. In view of the possibility and probability of iritis being due in whole or part to toxins, it was suggested that vaccines contain the soluble or exogenous toxins in addition to heat or chemically killed organisms.

Discussion. Dr. Edward A. Shumway spoke appreciatively of Dr. Kolmer's important communication. As a point of practical value in the treatment of iritis due to focal infections in various parts of the body, and especially those in the prostate, he said, that while gonococcal vaccine injections were most valuable where the gonococcus was present in the secretion after massage of the prostate, many cases were found in which there were no gonococci and only a mixed type of ordinary pus organisms. Such patients, according to urological specialists, are quite common and in them an autogenous vaccine was indicated.

After administration either of the gonococcal vaccine or of one prepared from the pus organisms present, the treatment should be followed by injection of one of the milk preparations, which will give a good nonspecific protein reaction. Such a case has been under his care recently and this method has been followed with marked success, with a perfect visual result.

DECEMBER 18

Roentgentherapy in Pituitary Tumors

Dr. George E. Pfahler and Dr. E. W. Spachman (by invitation) said that these tumors are of especial interest to the roentgenologist because by means of the roentgen rays one obtains some of the most valuable diagnostic evidence and because these tumors are the most satisfactory of all brain tumors for radiation therapy. Briefly, the diagnostic evidence depends upon some enlargement, destruction or deformity of the sella turcica which is produced by the pituitary tumor. Fortunately, these pituitary tumors, either sellar or suprasella, can be rather definitely localized, and since they are almost centrally located in a spherical body, the head, one can crossfire as much as is needed. As a rule, they are more radiosensitive than tumors elsewhere in the head.

We know that acromegalia is usually due to adenoma of the anterior lobe, while the dystrophy adiposa genitalis are usually due to suprasellar hypophyseal tumors, which are often indicated by lime deposit (Mackenzie, Sosman and Hirsch). In some cases, however, the location of the tumor is best indicated by a careful ophthalmological study.

The first case treated by the roentgen rays was reported by Gramegna, in 1909. A temporary improvement was reported and then ultimate failure. Béclère, in 1913, however, reported a case of a girl, aged sixteen years, in whom the vision improved threefold, the menses appeared, the breasts and pubic hair developed and she has been able to live a normal life since, for a period over twenty years.

The results from roentgentherapy are relief from the pressure symptoms, indicated chiefly by relief of headache and relief of the eye symptoms to a greater or lesser degree. Some cases do not respond at all. The cystic tumors seemingly are better treated by operation.

The eye symptoms show both objective and subjective improvement. The field of vision is increased, and a careful study of the fields of vision from time to time is the best objective means of recording progress or failure.

Since it is impractical to do a biopsy in these cases, and since the operation must be more or less formidable, it would seem advis-

able to use irradiation within the limits of normal tissue toleration during a period from six weeks to six months, under the observation of the ophthalmologist and neurologist, and if there is no response or insufficient response during this period of time, then it would seem advisable to do operation.

The presentation is accompanied by a description of the technique used and the results obtained in 5 additional cases, added to those reported by seventy-six other authors who have written upon this subject.

Discussion. Dr. Luther C. Peter said that Dr. Pfahler's reference to the value of this sort of therapy in some of these growths, and also his reference to the fact that when the tumors are in the pouch of Rathke the results are not quite so satisfactory, led him to mention 2 cases that came under his care during the last two years, in which operative technique started the patient on the road to improvement, but it remained for *x*-ray therapy to complete the cure, at least in 1 case and maybe in the other. As in most suprasellar tumors, in contrast to those below this level, choked disks were present in both instances.

One was the case of a young woman, aged eighteen years, with choking of the disk to the extent of 3 diopters. Operative interference improved the condition very much; headache, however, did not disappear, nor did the visual field really begin to fill out until she received deep *x*-ray therapy. This girl has made an absolute perfect recovery, if one can call it recovery at the end of two years. There are no symptoms other than an atrophy, which is very slight indeed. Her visual fields are full and she has no headache; in fact, she has no symptoms whatever.

The other case was a girl, aged twelve years. The symptoms were very severe, the onset really fulminating in character. The disks were choked to the extent of 6 or 7 diopters, not only in height but in breadth, so that the macula was invaded. There was total loss of central vision and the macular vision did not return. She was profoundly sick. She also improved after operative interference, but only after deep *x*-ray therapy was practised did the headaches really disappear and her improvement become very much more marked.

Dr. Adler said that, a few years ago, someone reported in the *American Journal of Ophthalmology* the case of a child with supposed pituitary tumor who fell from a porch and, in falling, landed on her head. After this fall all symptoms entirely disappeared. The author of this report supposed the tumor to be a cyst, which had been ruptured by the fall and a cure was obtained.

Dr. Adler said that at present they had in the Wills Hospital, on the service of Dr. Baer, a little girl with supposed pituitary tumor. Her only symptom is blindness. The disks show a postpapillitic atrophy. There is no evidence of increased intracranial pressure. There has been a com-

plete erosion of the sella into the sphenoid sinuses, so that the tumor has decompressed itself. Dr. Adler had seen the *x*-ray plates that day.

It naturally recalled the case referred to above and the possibility of the same thing happening there. Dr. Adler asked whether it is common to see things of this sort, *e. g.*, to have tumors of the pituitary erode into the sinuses? He also asked Dr. Pfahler whether he would recommend radium and *x*-ray therapy where the vision is deteriorating, due to increased intracranial pressure? He would like to know if it is justifiable to use radiation in such a case, or should one insist on surgical decompression?

Dr. Ring referred to some of the more recent literature upon pituitary body disease, and elaborated, at Dr. Pfahler's request, the case of a male patient, aged forty-nine years, who consulted him in April, 1928.

The general symptomatology included excessive sweating, abnormal sleepiness, pronounced lapses of memory and marked depression. The bones, especially of the hands and face, showed enlargement. The vision had begun to fail three years before examination. Tonsils, teeth and sinuses had been studied, but no consideration had apparently been given to the base of the skull. Vision, O.D., 20/50, and O.S., eccentric counting of fingers at 2 feet. The tension of each eye was within normal limits. No organic fundus changes other than those of the nerve head were apparent. Neither the rare manifestations of nerve choking nor inflammation of the disk was present. The "waxy" pallor of Benedict was not striking, but the disk in the left eye was slightly yelow and that of the right rather evenly changed in color and too pallid. There was no associated colloid goiter. The result of several examinations as regards glycosuria was negative. Bitemporal hemianopsia was present. At the end of forty-eight hours *x*-ray studies were made at the Graduate Hospital under the direction of Dr. Pfahler. Upon the receipt of his report, three days after the initial examination, Dr. Frazier was asked to examine the patient. After the first *x*-ray treatment givey by Dr. Pfahler, from the therapeutic viewpoint, his vision improved in the right eye to 20/40, one error. After the second application it improved to 20/30 and by May 7 to 20/20, four letters. By June to 20/20 all. The opposite eye could outline hazily 20/200. The striking change in O. D. has been preserved up to this time. The fields, as indicated by the charts shown at the meeting, also pointed to notable betterment.

Dr. Ring said that if he interpreted Dr. Frazier's letter correctly, the case represented the longest preservation of vision and field that he had seen as the outcome of radiation. No other treatment whatever had been given, and the man was so much improved that he was entirely disinclined to any operative interference.

The Surgical Treatment of Pituitary Tumors

Charles H. Frazier, M.D., Sc.D., read a paper on this subject, and he prefaced his remarks by calling attention to the enormous increase in knowledge of the pathology and symptomatology of

pituitary disorders since his first operation in 1912. At that time he was wont to be content with the comprehensive term "pituitary lesion," but now the pathology has become very complex and complicated. With the increased knowledge of the pathology and clinical expressions of pituitary disorder, the number of cases applying to the Neurosurgical Clinic has been steadily increasing, until at the present time 343 cases are recorded on the pituitary register.

Dr. Frazier addressed his remarks chiefly to the indications for surgical interference and the accomplishments of operative intervention. He reminded his audience that 97 per cent of cases sought relief because of failing vision; the remaining for intolerable headache. He called attention to the fact that within the last week he had operated for the first time in the absence of any failing vision. The patient, a young woman, showed early signs of acromegaly, and in order to prevent the progress of the disease with its resulting uncomeliness and unsightliness, he advised the patient to have the operation. This is the first case in which this operation has been done when the indication was not loss of vision. If eventually the sphere of indications for surgical interference is widened to include acromegaly or the Frolich syndrome, the activities in this field will be very much increased.

With regard to the question of intervention, the criterion now and always will be the presence or absence of optic atrophy and the degree of the same. Dr. Frazier reminded his audience that field distortion was a matter of secondary importance, as field distortion, due always to pressure or to blockage, might disappear within an hour of the operation. It was the presence or absence of optic atrophy upon which he relied mostly in expressing an opinion as to the possibilities of the individual case.

Of more importance and of more interest to the members of the Section is the statement as to the possibilities of surgical intervention. It is too soon to forecast what may be the results of operations for the arrest of acromegaly or other forms of dysfunction. With regard to headache the results of operation are inevitably satisfactory.

The crucial test, of course, as to the surgical efficiency with relation to pituitary disease has to do with vision. What may we expect as a result of operation? It all depends upon how much of the nerve or chiasm is atrophied at the time of the operation, and how far advanced the atrophy may be. As a matter of fact, almost

50 per cent of cases come to the clinic already blind in one eye, with rapidly failing vision in the other. Obviously it would not be fair to charge the operation with 50 per cent failures because vision could not be restored in these cases.

Generally speaking, Dr. Frazier said, the results may be put in three classes, all depending upon how little or how much atrophy there might be before the operation. Group 1: Vision may be fully restored. Group 2: Hemianopsia may persist, but vision in the nasal field may be restored or improved. Group 3: Hemianopsia may persist and visual acuity of the nasal field may not be improved, but may be sustained. These three alternatives supply the possibilities of surgery, and which of the three will maintain after operation is not a matter of surgical skill or surgical procedure.

With regard to the technique: This is a matter of little interest to the ophthalmologist. Suffice it to say that a technique has been elaborated which Dr. Frazier believes will be generally accepted. By this method, which he has been using for the past year, Dr. Frazier operated upon some 10 consecutive cases without a death. The minutiæ of the technique are not of general interest, but what is of interest to all is the fact that a method has been elaborated of approaching and dealing with the pituitary which has minimized the hazard of operation, and if operation be not postponed too long may restore vision to normal and maintain it.

Upon the conclusion of his remarks, Dr. Frazier showed a number of interesting lantern slides, illustrating the results in some of his cases.

The Ophthalmological Aspects of Pituitary Tumors

Dr. Holloway discussed the relation of certain vascular structures to the optic nerves, chiasms and tracts, and then referred to the various ocular manifestations encountered in pituitary body disease. A series of slides were shown, depicting the successive field changes from the superior temporal slant to the obliteration of the remaining field, which in his experience is usually in the superior nasal quadrant.

Attention was called to the necessity of repeated examinations in these cases, inasmuch as marked changes are frequently noted in short periods of time, independent of any treatment.

He stated that some of these cases were such as should produce a Wernicke pupillary sign, but he had never encountered it in this

or in any other condition, and he was one who believed that this sign could not be elicited by any of the convenient clinical methods now available.

Discussion. Dr. Frazier said he would like to make record in his series of pituitary lesions of 3 or 4 cases of what were called "pseudo-tumor;" that is, cases with all the earmarks of a solid tumor and with the characteristic field distortions. The pathological factor in these cases is a suprasellar collection of fluid, the evacuation of which suffices to give relief; 1 of these cases was under observation eight years.

PROCEEDINGS

OF THE

SECTION ON OTOLOGY AND LARYNGOLOGY

JANUARY 15

Observations on the Transillumination of the Frontal Sinuses

Dr. Edward H. Campbell: Transillumination of a frontal sinus will illuminate, not only the side on which the light is inserted, but also to some extent, the other side. The rather thick anterior wall of the opposite side transmits this light only faintly but the lower wall being thinner, transmits the light much better, and it shows as a dull glow at the upper inner angle of the orbit, opposite to the one where the light is inserted. When the glow is seen in this position, it means that the light has gone through three plates of bone; the lower wall of the sinus tested, the intervening septum, and the lower wall of the opposite sinus. Such a picture signifies clarity of both sinuses, as thickening of mucous membrane or collections of fluid in either sinus would prevent light transmission. In the case of large frontal sinuses such observations are unnecessary, as a clear outline of both sinuses is obtained. When one or both sinuses are small, failure to transmit light may be due to pathological change or to small air spaces. A glow under the opposite supraorbital ridge would indicate that both sinuses were clear.

When septa are present within the sinuses, when the bone is of abnormal thickness, or when the frontal sinuses are small, with considerable compact and cancellous bone between the two sinuses, neither sinus transilluminates well and the glow in the opposite upper inner angle of the orbit fails to appear. The above-mentioned conditions may then be suspected, unless the clinical history or the appearance of the mucous membrane in the region of the hiatus

semilunaris, as shown by the nasopharyngoscope, suggests a sinus infection.

Observing the absence or presence of this glow at the upper inner angle of the opposite orbit is an especial aid in the diagnosis of pathological conditions in the frontal sinuses of patients between the ages of four and fifteen years, in whom the frontal sinuses are small. In children between the ages of three and one-half years and eight years, transillumination, as usually observed, is of no value, because the frontal sinuses are not large enough to give characteristic illumination above the supraorbital ridges; but there can be observed in many cases, this glow of light at the upper inner angle of the opposite orbit, indicating that both frontal sinuses are clear.

Discussion. Dr. Samuel R. Skillern, Jr.: I have never observed the spot which Dr. Campbell refers to on the opposite side. Transillumination of the frontal sinus may fail, due to infected supraorbital cells. Polyps or pus in infected supraorbital cells prevent transmission of light in the case of a perfectly healthy frontal sinus. Transillumination of a diseased sinus by diffused light under the soft tissues is possible. Roentgenograms are superior to transillumination. If the roentgenogram disagrees with your diagnosis, however, it is probably wrong. With symptoms of frontal sinus disease and transillumination indicating a black frontal sinus, frontal sinus disease exists even if the sinuses are negative by *x*-ray. In one case with symptoms of right frontal sinus involvement, we desired to do a radical operation. Two roentgenologists, independently, reported absence of the right frontal sinus. I put a probe in the right frontal sinus and Dr. Widmann took plates showing the probe in the sinus. At operation the bone was found to be $\frac{1}{2}$ inch thick.

Dr. Philip Stout: We get too close to the nose when we transilluminate. I think this is what we see. I never remember seeing light go through to the opposite side if we go out far enough from the midline.

Dr. Campbell: I do not think there is any question about this light going through. The combined thickness of the septum and the lateral wall of the nose is greater than that of the frontal septum and the inferior wall of the frontal sinus. The thickness of the skin and the subcutaneous tissues over the forehead is several times thicker than similar structures in the orbital area. This method is not infallible. I do not believe the ethmoidal cell mentioned by Dr. Skillern is a factor in this transmission of light.

Dr. Warren B. Davis: The illustrations Dr. Campbell has presented speak for themselves. They are anatomical facts. The reason he gets these results is because of the type of transilluminator he uses. It has no lateral radiation and therefore should pass through the structures as he describes. I have not used that type of transilluminator and therefore did not feel qualified to discuss it.

Some Practical Points in the Use of Electrosurgical Measures in the Upper Respiratory Tract

Dr. Marshall B. Sponsler: After reviewing the use of high frequency electric currents during the past twenty years, Dr. Sponsler emphasized the purposes to which the three modalities in use at present are best adapted. In the treatment of nose and throat conditions, the D'Arsonval current is used, with a large or indifferent electrode, placed over the mid-dorsal region, and a small active electrode, varying from the size of a large darning needle to a small sewing needle. The small electrode, inserted into the tissue for a distance of a $\frac{1}{4}$ inch, causes the tissue to coagulate to a greater or lesser depth, depending on the strength of the current and the length of time of application. Cutting is accomplished with a current whose to-and-fro movements are as many as three millions per second. The cut surface of this divided tissue appears seared and wounds of this sort, when sutured, heal by first intention.

The small electrode may be merely a tip or loop-shaped, the latter being useful in removal of bits of tissue for diagnosis. The combination of snare and cutting or coagulating current is of advantage in removing very vascular growths in the nasal cavity.

The proper setting of the machine is very important and a foot switch is absolutely necessary so that the current is under control of the operator at all times. Small growths and superficial lesions of the nose and throat may be anesthetized with 10 per cent cocaine solution or 1 per cent novocain solution. Larger growths involving deeper structures demand general anesthesia. Gas or chloroform should be used rather than ether, which is inflammable.

For tonsil destruction the machine should be so set that a snow-white circle of coagulation with a diameter of $\frac{1}{4}$ inch is produced with one second application of the current. Longer application produces charring and a sparking effect. When mucous membrane is coagulated, considerable charring with hyperemia and edema results. The mucosa of the nose is very sensitive, while the tonsils are least sensitive, in some instances application being possible without a local anesthetic.

Secondary hemorrhage may be serious when coagulating lesions in the vicinity of larger bloodvessels and proper ligation should be advised. Other untoward results are due to faulty technique as: (1) Accidental contact of the active electrode with other parts

than those treated, and (2) Faulty contact of the indifferent electrode resulting in burns. After electrocoagulation of the tonsils, there is not sufficient pain to cause the patient to refrain from eating and none complained of any feeling of illness. There is practically no pain during the treatment, and while pain occurs during the two or three days following a seance, in about one-fourth of the cases no pain or discomfort was noticed at all. Pain in the ear on the side of application appears almost constantly when treating in the region of the capsule in the middle of the tonsillar fossa and the upper pole. No bleeding appeared during any of the treatments but occurred twice in the same patient five days after coagulation. This was not of a serious nature. There is no evidence of toxemia after such treatments. The tonsillar fossæ, in which the tonsils so far have apparently been removed, present a smooth surface, covered with epithelium, with no evidence of dense, white scar tissue. The healed area is soft and pliable. Since an average of 18 treatments was given each patient, it is a time-consuming method for patient and physician.

Discussion. Dr. George B. Wood: I have been interested in trying out fulguration in tonsillar tabs and small tonsils. The lack of discomfort is noteworthy, in contrast to the results after the actual cautery. The fulguration method coagulates and desiccates. I am disappointed that the method does not destroy the tonsil as fast as you would expect it to. I am told that fulguration is easier to control in inexperienced hands than electrocoagulation.

Dr. Fielding O. Lewis: I believe this method is indispensable in cancer work. It is useful where part of the tongue or like structures must be removed. We should always be equipped by cutting current as well as by the electrocautery, in case the extent of the cancer is more extensive than we supposed. I agree with Dr. Sponsler that it does not replace surgical methods but is useful when contraindications to surgery exist, such as bleeders or patients fearful of surgery. Where we have used the method, the final results were satisfactory.

Dr. S. R. Skillern: Two years ago I bought a machine and have done 38 cases. Everything Dr. Sponsler has said is true. I want to emphasize one thing, which I feel should not be overlooked. If the time between the treatments is lengthened for any cause, the anterior and posterior pillars have a trick of closing across the tonsillar fossa. In one case the tonsillar fossa had almost closed over a large piece of tonsil and free pus could be expressed on pressure. The patient had had systemic symptoms of a focus of infection for ten days. The removal of tonsil tissue by electrocoagulation is more difficult than by the surgical method. The methods have been misrepresented by osteopaths, naprapaths and others and there will be a lot

of ruined throats. We will see pillars grown across and foci underneath. After sufficient tonsil tissue is removed, the residual tissue slips behind the anterior pillar. In coagulating this tissue it is easy to slip through to the anterior pillar and then tremendous edema with pain and reaction follows. This is simply due to touching muscle tissue. If the uvula is touched it gets as big as two thumbs. The sale of these machines should be restricted so that they do not fall into the hands of the unscrupulous.

Dr. George M. Coates: In 1915, Leslie Mulford and I tried a fulguration machine on cases not suitable for surgery. We only attempted surface coagulation. Two years ago I tried the method used by Dillinger. Of the cases I have done recently, there is an uncertainty as to the completeness of tissue removal. The electrotherapeutists remove tonsils in one or two sittings. In three of these, my hospital assistant has been called in to stop hemorrhage. It is very difficult to stop hemorrhage from this source. I have been told that pillars do grow over infected tonsil tissue in these cases and when this occurs, the condition is worse than it was before.

Dr. Sponsler (in closing): Medical opinion kept me from trying this method earlier. Novak of Boston reported a case several years ago, in which repeated examinations by capable otolaryngologists failed to reveal residual tonsil tissue in such a case, because contraction of the pillars had occurred after coagulation. I do not know that there is an inclusion of tonsil tissue but can see that it would be difficult to discern this tissue. At first the Germans and French tried to take out a whole tonsil at a sitting. This was a system of coagulate and curette, until the capsule of the tonsil was reached. This of course should be a discarded method. Two years ago I saw a woman who had her tonsils removed at one sitting. A small tab remained. She had had so much bleeding that she was in bed for a week. I later had no difficulty in removing the tabs so that she could not have been a bleeder.

Two Cases of Brain Abscess Presenting Some Interesting Features

Dr. Austin T. Smith: The first patient, a male, aged thirty-three years, had had an acute otitis media, which cleared up promptly after myringotomy. A few weeks later, the patient became dull. lethargic and stuporous. Pain centered over the right frontal area. At this time there was no evidence of mastoid disease, the temperature was normal and the pulse slow. There were 74 cells per cubic centimeter of spinal fluid, 93 per cent being lymphocytes and 7 per cent polymorphonuclears. Neurological opinion favored the diagnosis of encephalitis and he was treated on this basis for four weeks, during which time a low-grade neuritis developed on both sides but was more marked on the left. Pain was persistent over the frontal region. During the sixth week, weakness of the right third nerve

and of the left seventh nerve was noted. The eye-grounds showed optic neuritis with hemorrhage. There was a swelling of $1\frac{1}{2}$ diopters on the right and 3 diopters on the left. The right mastoid area was opened but the cells gave no evidence of inflammatory change and the inner table was intact. An opening in the inner table was made but no lesion discovered beneath. Forty-eight hours later, weakness of the left arm and leg without spasticity or clonus were noted and the patient became stuporous. The right temporoparietal region was exposed and the temporal lobe explored with a needle. A very large abscess was found and a large quantity of pus aspirated. A Type IV pneumococcus was isolated in cultures. Ten hours after operation, the patient died, although he had improved markedly immediately after the removal of the pus. Few cases have been reported in which healing had occurred and the perforation of the tympanic membrane had closed prior to the onset of other symptoms.

The second patient, a coal-miner, aged twenty-nine years, had had "catarrh" of the nose since childhood and polypi had been removed from his nose on several occasions. While otherwise in good health, pain had appeared over the left forehead, usually in the morning and lasting a few hours. Pain increasing in severity, and redness and swelling appearing over the left frontal and temporal areas and over the upper eyelid, the patient was admitted to the hospital. At this time the temperature was 102° F., with a pulse-rate of 100, and respirations of 20. The neurological findings were completely negative. Infection of both frontal sinuses and both ethmoidal sinuses was reported by the roentgenologist. A radical Killian operation on the left frontal sinus revealed foul-smelling pus in the sinus. The lumen was filled with polypoid tissue and hyperplastic membrane. A communication with the right frontal sinus revealed a similar condition there. Both frontal and both ethmoidal sinuses were exenterated and free drainage established into the nose. The inner table of the frontal bone was intact. Improvement followed the operation but later symptoms of bronchopneumonia appeared and death ensued seven days after operation. Postmortem examination revealed a large abscess occupying the whole of the anterior portion of the left temporal lobe. The dura was densely adherent to the skull in the frontal region but there was no evidence of inflammation on its inner surface. At the base, in the left middle fossa, small areas of the dura were inflamed and

necrotic. No suppurative tract led to the sinuses and there was grossly no evidence of the path of infection from the sinuses to the brain. When the sphenoidal sinuses were opened, they were found to contain pus. Because of the close relationship of the anterior part of the temporal lobes to the sphenoid bones, it is quite likely that the infection of the brain came from this source, probably by means of a retrograde thrombophlebitis.

Discussion. Dr. Francis C. Grant: I have great diffidence in discussing brain abscess because I have great sympathy for the difficulty of diagnosing these lesions. In these cases where an attempt is made to localize an expanding lesion, all potentialities that might converge to tumor should be applied to the problem. I feel that careful perimetric plotting would have aided localization in the first case. Quadrate defects should be looked for in such instances. I would suspect an abscess first in the presence of such nasal disease in spite of the neurological opinion. When in doubt, the neurologists are too prone to fall back on the diagnosis of encephalitis. Increasing stupor and slow pulse (the latter certainly not typical of encephalitis) would indicate abscess. I would not have hesitated to explore the temporal lobe to eliminate the possibility of abscess. I was interested in the question of doing osteoplastic flaps for brain abscess. From the neurosurgical standpoint, this is the last thing the neurosurgeon would do because this opens up a large field. If the patient dies, it is due to too much interference resulting in cerebritis. We feel that small trephine openings and the smallest drain should be introduced, the drain allowed to remain so that the abscess is permitted to drain and collapse slowly. If the drain comes out there is no need to worry. We have done harm in being in too much of a hurry to get that pus out. These brain abscesses are extremely protean in their manifestations. We must exhaust all our resources to eliminate the possibility of brain abscess in such cases. If in doubt, a trephine opening should be made and plunge through a sterile field. If we do not find the lesion then we may follow along with our neurological friend and consider encephalitis. The proper procedure is to wait until the abscess is well encapsulated and localized. Then drainage may be safely done without spreading infection from the drainage tract.

Dr. Benjamin H. Shuster: In this case the mastoid was opened, trephining done and the dura opened after paralysis of the right third nerve and the left seventh nerve. In face of the depressed mentality and the localizing symptoms afforded by the nerve involvements, the brain should have been explored. Macewen has pointed out that the nuclei of the third nerve are situated medially and deeply within the gray matter of the floor of the sylvian aqueduct and are the first involved. Therefore the procedure should have been to open up at that time.

FEBRUARY 19

Vasomotor Rhinitis

Dr. Harold G. Tobey (by invitation), Boston, Mass.: A wide divergence of opinion exists as to what clinical symptoms constitute presumptive evidence of vasomotor rhinitis. Since sneezing, rhinorrhea and nasal obstruction are the outstanding symptoms of vasomotor disturbances of the nose, a definition of these symptoms defines vasomotor rhinitis. Articles dealing with rhinitis, hayfever, nasal hydrorrhea, paroxysmal hydrorrhea, hyperesthetic rhinitis, atopic coryza and allergic rhinitis describe a vasomotor condition of the nose the exact nature of which is not definitely defined, and frequently the thesis becomes a frank discussion of true seasonal hayfever or the relation of such nasal conditions to asthma. Dr. Tobey prefers the term vasomotor rhinitis because rhinitis suggests an inflammatory reaction. Many cases, of course, do not show typical acute inflammatory change. Perennial coryza is a good term but is newer and less familiar than vasomotor rhinitis.

At the time Dr. Tobey became associated with the clinic as a rhinological consultant, the cases were put through a tedious siege of skin tests. Frequently the tests were all negative and at other times cases with positive reactions failed to improve under treatment. Dr. Tobey found that while the symptoms in different individuals were often alike, there was a difference in the appearance of the nasal mucous membrane. In the vasomotor cases that were allergic the membranes were apt to be pale, while in nonallergic cases the membranes were red and beefy. The allergic side has progressed a great deal and many associations and a journal are now interested in diffusing information on allergic problems.

Vasomotor rhinitis may be divided into the allergic and the nonallergic types. A clear idea of what is meant by the term "allergy" is essential for a rational approach to this subject. Some neurological diseases, such as angioneurotic edema or epilepsy, have been connected with allergy. Gastrointestinal and bladder symptoms may be less characteristic. The history is of great importance and the patient's observations are frequently important. Of the specific clinical tests, skin reactions have received the most attention, but they are by no means infallible. Occasionally the skin reacts to but one substance and if that substance is rare we may expect good

results to follow its removal. Ophthalmic and nasal tests are also an aid in obscure cases, especially where we are dealing with dusts and pollens. The diversity of substances which may be a factor is evident when we consider that orris root, smoke, dust, foods, drugs and bacteria as well as the skin, roots and seeds of plants have frequently been shown to be etiological factors. Insects, animal parasites, therapeutic serum, light, heat and cold are factors and pregnancy, menstruation, nasal defects and functional disorders of the alimentary tract may influence the reactions.

Duke and Kyle point out the pale swelling which is so characteristic of vasomotor rhinitis, but it is not a common statement. The sneezing and pale swelling are associated with a clear, watery secretion, but the former may occur without a pale mucous membrane. Secretion may vary from mere moisture to the outpouring of pints. Sinus obstruction and pain may result from the obstruction of the turgid turbinates. When it is associated with asthma we usually suspect an air-borne substance and the chronic edema of air-borne cases frequently results in polypi. The duration of edema in the seasonal cases is too short to give this result. It is necessary to rely on the family history and the personal history and depend on the finding of some substance to which the individual reacts to make the diagnosis.

Nonallergic cases may be due to extrinsic or intrinsic causes. Numerous illustrations of extrinsic causes occur in all occupations and especially in rubber industries, shoe and chemical workers. Direct irritation or reflex irritation may be due to factors within the nose itself (infection in the ethmoidal cells, swollen turbinates, deviation of the septum, infection in the frontal and maxillary sinuses).

The vasomotor rhinitis cases due to direct irritation are the ones which are likely to benefit from surgery. In one case where skin tests, endocrine therapy, cauterization and roentgen-ray had been tried without improvement, exploration of the right antrum disclosed 2 drams of straw-colored fluid. The ostium was extremely small, being almost pin-point in size, with the mucous membrane immediately surrounding it definitely congested. An immediate cessation of all symptoms followed and the relief has persisted for seven years.

Another case, that of a professor at Harvard, was characterized by sneezing and great rhinorrhea. The roentgenologist reported

diffuse cloudiness of one antrum, but it transilluminated well. On aspiration a cyst was found and emptied and in twenty-four hours all symptoms had disappeared. Upon the recurrence of symptoms, a radical antrum operation was done and a good part of the mucous membrane removed. There has been no recurrence for two years.

In the reflex irritation group must be included etiological factors such as teeth, especially unerupted third molars. Dr. Sluder's method has been used in some reflex cases but this has not been successful in my hands. In one case so injected severe hemorrhage was encountered, so that thereafter I used the palatal route to reach the sphenopalatine ganglion. Three of such cases were benefited and 3 other cases failed to return for follow-up.

Are the endocrine cases allergic or nonallergic? Theoretically they should be in the nonallergic class. Four of these cases had pallor of the mucous membrane while 2 showed no pallor, and of these 6 cases, 5 were ovarian and 1 pituitary. The pituitary case was unquestionably allergic. The pituitary gland may have had little to do with it but the administration of pituitary extract was followed by improvement. This patient's father was an epileptic and her mother had asthma. The attacks began with migraine headaches followed by typical vasomotor rhinitis and later asthmatic wheezing. After many operations and removal of the tips of the turbinates the pituitary extract was administered and relief obtained. Another woman whose symptoms had started two years before at the time of a tubal pregnancy and hysterectomy was immediately relieved when ovarian extract was administered. Withdrawing ovarian extract immediately was followed by recurrence of symptoms and this could be stopped by giving extract.

Dr. Tobey agrees with Dr. Fink's classic description of the general pathological picture observed in these cases and also with his theory of polyp formation. Dr. Tobey's rule for the past five years has been to consider surgery in the true cases with reddened mucous membrane. The red mucous membrane after seasonal hayfever frequently continues through the rest of the year. In these cases submucous resection or the removal of a bulbous turbinate benefits the allergic case. Antrum infection is also important here.

In pale, swollen cases surgery is the very last thing to attempt. Cocaine will not shrink the tissues. Polypi may be removed (true allergic polypi) and they will return.

At first these cases were referred to the allergists. Now allergy

has outgrown the otolaryngologist. Allergy has become a specialty, and rightly so. However, the rhinologist should investigate his cases and ascertain whether they are allergic or not. The pendulum seems to be now swinging the other way and we find the allergists are sending such cases to the otolaryngologist. Both investigators must work together and much remains to be learned about therapy, for the treatment of these patients is more unsatisfactory than that of asthmatics.

Discussion. Dr. Richard A. Kern: There is little that I can say by way of amplification and as an allergist I am a bit one-sided. The cases that I see have been referred by the otolaryngologist. I agree with Dr. Tobey's classification of the changes in the nasal mucous membrane. Such a classification has been of value in certain obscure cough situations. Bronchoscopic studies have revealed similar changes in the bronchial mucous membrane and the pallid cases have frequently proven to be allergic. In ferreting out the causes of these situations, the history is of the greatest importance, and there is no history more difficult to take. All ramifications must be considered—family history, climate, occupational contacts, diet, etc. Not only the obvious allergic factor but all other allergic manifestations must be investigated.

The interpretation of skin tests is of vast importance. A strongly positive reaction may indicate allergy but not absolutely. We must have clinical proof as well. The substance must induce attacks and its withdrawal should stop attacks. Moreover, skin sensitivity sometimes does not mirror the sensitivity of the rest of the body. Ragweed hayfever may occur without skin or conjunctival reaction, but relief will follow treatment with pollen extracts. Frequently patients will give a history of extensive nasal surgery without relief. These are probably purely allergic cases. The degree of sensitivity may depend on the permeability of the nasal mucosa. An irritable state of the nasal mucosa, the result of contacts, etc., is accompanied by congestion which will make the mucous membrane at that point more permeable. Therefore, there is more absorption at that point. This is not only hypothetical. An individual may be sensitive to a pollen and to goose feathers. His symptoms may occur only during the pollen season, but the administration of pollen extracts will not cure him. If we now remove the additional factor (goose feathers), a brilliant therapeutic success is obtained. At other season the mucous membrane is not sufficiently permeable to the feather factor. Upper respiratory infections may produce a similar phenomenon.

I would like to ask Dr. Tobey what percentage of the vasomotor rhinitis cases are of the red mucous membrane type and of the pallid types? As a result of the congested and edematous change polyps do arise. Clinical and roentgenological examinations of the sinuses are of importance. Surgery should be the last consideration in treatment of the pallid mucous membrane cases. Having found an allergic basis, it is often surprising how often a blocked sinus will drain of itself. I have often wondered why more

pituitary and adrenal cases have not come in because extracts of these substances frequently are efficacious, and I have wondered why functional disturbances of these glands have not been described.

Dr. H. P. Schenck: I have always felt indebted to Dr. Tobey for his publication in 1925 of the neurological factors involved in vasomotor rhinitis. He has also performed a valuable service in first pointing out the characteristic appearances of the nasal mucous membrane in such cases, thus bringing diagnostic order out of speculative chaos. Considerable aid in diagnosis may be afforded by the cytological study of the secretions in these cases. Eyermann has described eosinophils in such secretions and I wonder what difference the secretions in the reddened type exhibit that the pallid type do not have. A simple smear stained by Wright's method would be sufficient. The pathological studies of such tissues described the eosinophilic increase in the tissues, especially in the antra in asthmatics. Here, the greatest collection of eosinophils appears near the mucous membrane. The increase of intercellular fluid has been noted while the vascular channels appear unchanged. In cases of greater duration polyp formation is the result of constriction of the venous channels by the weight of the edematous tissues, thus establishing a vicious cycle. Dr. Kern and I have recently studied the changes that occur in living preformed tissues in lower animals under the stimulus of allergens. Here the tremendous increase of intercellular fluid, probably serum and lymph, is startling. Its rapidity of appearance is difficult to conceive. In spite of the other tissue activity, the vessels do not dilate but there is activity on the part of the arteriovenous anastomoses. Cytological observations are of especial interest in such preparations. The Rouget cell, for instance, has been observed to become active and to approach vessels in its vicinity and apparently partially enfold the vessel. These phenomena are hard to explain with our limited knowledge. More observations on living tissues are required to advance our conceptions of the mechanism of allergy.

Dr. F. A. Murphy: What we need is something which will help us separate the allergic from the nonallergic individuals. An extract of human dander has been used to differentiate allergic from nonallergic individuals. I was interested in Dr. Tobey's differentiation between the reddened and the pallid types. I see these cases from the allergic standpoint and therefore am a little one-sided. I have 2 or 3 cases which were sensitive to a definite substance as shown by ingestion. Removing the substance from the diet was followed by disappearance of symptoms. One patient was later told to eat eggs because of a run-down condition and the symptoms immediately reappeared.

Dr. Philip Stout: Regardless of whether a case is allergic or nonallergic, investigate the antra. In a surprising number of cases one or more polyps will be found. Their removal will give temporary or permanent relief.

Dr. Tobey (in closing): I was interested in Dr. Kern's remarks on bronchoscopic observations. I have not done a great many of these cases. I am not sure if this pallor extends down into the bronchial mucous membrane or not. Most of those that I have seen were due to bronchiectasis. The

relative number of pale and reddened mucous membrane cases is difficult to estimate because the mucous membrane does change. It may be red one time, white another or may show the pallid change in only a very small area. Surgery in these cases should only be attempted when everything else fails. I have not seen any of the adrenalin cases. The bony changes are not well recognized but they have been described in Dr. Beck's book. Dr. Murphy brought up Van Leeuwen's observation with human dander. I understand this is very difficult of corroboration. The antra are often washed as a matter of routine in our clinic.

MARCH 19

Symposium on the Relation of Sinus Disease to Pulmonary Infection, from the Standpoint of the Internist

Dr. Thomas McCrae: It is essential to have the coöperation of men in the various departments of medical science in order that a comprehensive grasp of this subject may be had. From the standpoint of medicine, it is well to go back and consider infections of the lower respiratory tract other than tuberculosis. This knowledge was gained gradually, and, I take it, the development from the otolaryngological standpoint was also a slow one. Inhalation must play a part; drainage a large part; lymphatic transmission has its importance; blood transmission is responsible for a very small amount.

The changes in the lower respiratory tract due to infection from above are, first, bronchial infection; later, lymphatic infection. The infection is at first acute, and there are periods of freedom. With more exacerbations, fibrosis occurs—a matter of years in most cases, but occasionally of rapid development. Cough results in emphysema, which increases discomfort. Bronchiectasis finally results. Sinus infection is probably the most important etiological factor in bronchiectasis. Lymphatic drainage may carry the infection. In one patient one change predominates; in another, another. Conditions are different as seen in children and as seen in adults. We frequently see a condition in children which is secondary to sinus disease. There is a continued fever, not high but persistent, and there is little in the respiratory tract to explain the fever. Pyelitis, etc., may be eliminated. The lymph nodes of the mediastinum are found enlarged. In this group the responsibility of sinus infection should be kept in mind.

Sinus disease should be considered when bronchitis in children assumes one of two aspects. There are repeated acute attacks with periods of freedom between attacks, but a tendency to chronicity. Coughing occurs during the winter, but disappears during the summer.

Of particular importance is the question of bronchiectasis. We have learned to recognize the full-blown picture of bronchiectasis. It begins in childhood, however, and at this time is not recognized. In asthmatics, sinus infection is of more importance in adults than in children. The glandular reaction is different in adults and in children. A brilliant result was obtained recently in one of my cases with a history of years of fibrous bronchiectasis and asthma. Although there had been no symptoms of sinus disease, the roentgenogram revealed it and treatment directed toward the sinuses had astounding effect on the pulmonary condition.

As far as diagnosis in general is concerned, we must always think of sinus disease and follow it up. Here teamwork is always very essential. Follow the question up and settle it, "Yes" or "No." On the other hand, we have the right to ask the best possible diagnosis. Sometimes the clinician is caught between opposing opinions.

It is important to differentiate the condition from tuberculosis. Many lower respiratory infections are secondary to sinus disease and are erroneously diagnosed tuberculosis. At an army hospital where I gave a course, we carried out an intensive campaign to find patients diagnosed as tuberculous who did not have pulmonary tuberculosis but sinus disease. We found a great many and this brought the lesson home to me.

There is perhaps no more dreadful disease than bronchiectasis. We should strive to recognize cases early and prevent it. If we investigated the sinuses oftener, we would help many. While the diagnosis of bronchiectasis is sometimes difficult, we should be suspicious of it because of peculiar râles, never heard in any other condition. The early recognition of bronchiectasis and the efficient study of the source of infection will prevent many of these cases. Bronchial asthma is a matter for teamwork between the roentgenologist, otolaryngologist, allergist and clinician. The clinical picture often gives the clue. You have the right to ask us to diagnose these conditions earlier, and we have the right to ask you to increase the accuracy of examinations. Thus we will separate tuberculous cases from the conditions which may closely resemble them.

From the Standpoint of the Rhinologist

Dr. William V. Mullin, of Cleveland, Ohio: The nasal sinuses do not play a direct part in the production of lung abscesses unless the secretions are inspired or unless a certain type of sacculated bronchiectasis is confused with lung abscess. I do not believe that they play a part in the causation of chronic daily elevation of temperature in either children or adults. They have only an infrequent and coincidental connection in pulmonary and hilar tuberculosis. Acute laryngitis, tracheitis and bronchitis are merely a part of an upper respiratory infection in which the mucosa of the sinuses may or may not be involved. The conditions in the chest that are affected by disease of the accessory sinuses are cough, bronchitis, bronchiectasis and bronchial asthma. In my experience, some of these conditions are the most outstanding complications of chronic sinus disease; yet I realize that this statement does not coincide with the opinions of many others.

In the past twelve years it has been my good fortune to have all my sinus and chest films read by but two roentgenologists, with whom I have been privileged to see the majority of the plates. The term "good fortune" is used advisedly, since the intuitive perception that enables a roentgenologist to differentiate between the markings indicating tuberculous and nontuberculous infections in the chest caused by sinus disease, can be developed only after he has followed the clinical course of a large number of cases in constant coöperation with the rhinologist.

A very careful and detailed history must be taken to bring out both general and local symptoms, and in every patient with chronic cough and bronchial asthma the sinuses should be considered as one of the causative agents until they are ruled out. The part played by the sinuses differs in the production of chronic bronchitis and bronchiectasis and the development of bronchial asthma, as the constitutional reaction and pathological factors are not the same in these conditions. The seed of chronic bronchitis and bronchiectasis is planted in childhood. It is the child that is susceptible to infections and suffers from frequent colds who is later afflicted with these chronic lung infections. Environmental influences and diet may be added factors, but not necessarily so. It is the continual dropping that wears away the stone. It takes years to produce bilateral bronchiectasis. The nasal and sinus membranes may never

recover entirely from congestion and edematous swelling between acute exacerbations during the winter months.

In sinus infection, while one sinus may be more involved than the others (and I believe the maxillary sinuses are the most important), the condition should be considered, so far as absorption from the infected mucosa is concerned, as a pansinusitis. Eleven years ago I demonstrated that the route of lymphatic absorption from the nose and sinuses is by way of the submaxillary and internal jugular nodes to the lymph ducts, the great veins, the right side of the heart and the lungs, then into the general circulation. It is still my belief that the bronchial, peribronchial and mediastinal nodes become infected through the blood and lymph route from the sinuses. The peribronchial glands extend down into the third and fourth orders of the bronchi, and enlargement of these glands will cause pressure in the finer bronchi, produce irritation and cough, interfere with bronchial peristalsis and cause retention of secretion with, eventually, a dilatation of the finer walls.

The question is often asked why some patients who have had chronic sinus infection have not developed secondary chest conditions, while others have marked chest symptoms. This may be explained on the basis of individual idiosyncrasy or some special type of infection, although the latter seems improbable. It might also be explained by morphological irregularities.

Bronchial Asthma. If it were possible to study from every angle over a long period of time a large group of cases of all types, definite information as to etiology and treatment would surely result. Unfortunately, the fact that a hypodermic of adrenalin gives prompt relief in a large percentage of cases has seemed sufficient, and not much effort has been made to learn the reason why. Allergists approach the subject from too narrow an angle, just as the rhinologists formerly did. Sinus surgery is abandoned before thorough correction is accomplished, and then criticized because the results are not brilliant.

The asthmatic is in an unbalanced condition, and if it is true that sympathetic nerve fibers can be demonstrated in the sinus linings, certainly a nasal reflex cause is comprehensible. The asthmatic state is constitutional, and the pathological condition of the mucosa of the sinuses may be, and undoubtedly is, a reflection of this constitutional condition. Yet I have seen many cases in which no cause other than infection in the sinuses could be found to

account for the asthma, and in which nothing was done aside from ridding the sinuses of infection, with satisfactory results.

Symptoms secondary to chronic sinus infection should influence the treatment instituted. Early recognition of sinus infection in children, with prompt treatment, should prevent many chronic chest conditions. In every case of chronic sinus infection with a cough, a complete roentgenologic examination of the chest should be made. The reverse of this is also true. After the bronchial tree is infected, general as well as local treatment must be carried out. Conservative treatment of the sinuses plus general care may be sufficient. However, if the sinus mucosa is diseased beyond its ability to recover function, operative treatment is required. In asthma, irrespective of allergic reactions or environmental influences, if the maxillary sinuses and ethmoids are definitely polypoid, only the most thorough removal, with repeated attempts to eliminate all infection, will produce satisfactory results.

From the Standpoint of the Bronchoscopist

Dr. Robert M. Lukens: Certain chronic diseases of the lung, such as bronchiectasis, chronic bronchitis and bronchial asthma, are associated with paranasal sinus disease. The sinus condition and the bronchial condition are concomitant rather than one being the cause of the other. This is probably true in the very early cases, particularly in children who have repeated attacks of so-called "cold in the head" and "cold in the chest." These tend to clear up under medical treatment, and the patient is supposedly cured. Later in life some of these children develop the clinical picture of bronchiectasis. The "cold in the chest" really did clear up, but the "cold in the head" remained as an infection of the sinuses, either without symptoms or with a discharging nose. A constant postnasal discharge into the bronchi may be established in conjunction with lymphatic drainage to the peribronchial tissues. Constant reinfection tends to a chronic bronchitis, which may cause weakening of the bronchial walls, and upon the advent of whooping-cough, pneumonia, influenza, etc., a dilation of the weakened bronchial walls will occur with the later development of bronchiectasis.

In the bronchoscopic clinic the great majority of the bronchiectasis cases have an associated sinus infection, which a requested rhinologic or roentgenologic examination brings to light. It is impos-

sible to say that sinus and bronchial infections are always associated, as there are many cases of sinusitis without suspected bronchial infection. To determine this matter, every case of chronic sinus infection should have a physical and roentgenological examination of the lungs, bronchoscopy being employed in cases definitely indicated.

In children with chronic cough and nasal discharge, before tonsils and adenoids are accused of being the only offenders, thorough *x*-ray studies of the lungs and sinuses should be made. In this stage of potential bronchiectasis there is hope of combating actual bronchiectasis. Bronchoscopic treatment has its greatest problem in the advanced cases with numerous cavities, particularly in older children and adults. These cases are very disappointing from a bronchoscopic standpoint. The earlier the treatment, the greater the success. After the bronchial walls are damaged, no amount of bronchoscopic drainage will restore them to normal. In addition, thorough drainage of the sinuses must be carried out.

Where bronchoscopic drainage was employed in asthmatics, after the removal of polypi and sinus infection, almost miraculous results were obtained. On the other hand, cases without previous rhinologic examination and treatment did not improve after bronchoscopy. The removal of infection from the sinuses was half the treatment. Not all cases of chronic bronchitis or early bronchiectasis associated with sinus disease must be treated bronchoscopically. Quite a number referred to the rhinologist will lose all bronchial symptoms under proper sinus treatment. These are cases in which the bronchial disease has not reached the stage where structural damage has occurred in the bronchial walls, and after overflow from the sinuses ceases the ciliated epithelium can take care of the bronchial infection. In older cases the ciliated epithelium is destroyed and mechanical assistance is necessary to remove stagnant secretion.

Pneumonography is employed to make the bronchi opaque. The bronchi are filled with iodized oil or bismuth subcarbonate powder. This is best done by the bronchoscope, since this lessens the danger of carrying infection from the mouth into the lung and the preparation can be more accurately placed. An opportunity is also afforded to study, visually, the bronchi and to obtain a specimen of secretion for microscopic study. The bronchi may be made to stand out as empty tubes in the *x*-ray film by insufflating bismuth subcarbonate powder through the bronchoscope, some of which will adhere to the bronchial walls while the excess is coughed out.

The bronchoscope is absolutely essential to make a differential diagnosis between simple chronic bronchitis associated with sinus

disease and other lesions of the bronchi which may exist coincidently with sinus and bronchial infection. The presence of congenital webs, compression stenosis produced by peribronchial or peritracheal glands, cicatricial stenosis, polypi, new growths, granulations, etc., can only be accurately diagnosed by bronchoscopy. A diagnostic bronchoscopy is essential in asthma associated with sinus disease, chiefly to determine the degree of inflammation and character of the secretion, the possibility of obstruction to drainage, and to obtain material for vaccine.

Bronchoscopic drainage may not be necessary in early cases of sinus and bronchial infection before bronchiectatic cavities are formed. If the cilia of the bronchial epithelium have not been hampered or destroyed by stagnant secretion, treatment of the sinus condition may result in clearing the bronchial infection. Later, when the bronchial mucosa has been damaged to such an extent that ciliary action is unable to raise the secretion to the point where it may be removed by cough, it is necessary to remove the secretion mechanically. The mechanical removal is repeated until the mucosa has recovered. In advanced cases of bronchiectasis, with numerous large sacculated cavities, bronchoscopy from the standpoint of cure is ineffectual. This is the fault of delayed diagnosis, for, by the time large cavities have formed, the tissues of the bronchi have been destroyed and return to normal is impossible. Regular bronchoscopic drainage will, however, remove the decomposing material, lessen or abolish the terrible odor and give the patient comfort for varying periods of time. Asthmatic cases are usually well established before coming to the bronchoscopist. The secretion is often semitransparent, tenacious and is removed with some difficulty, Epithelial cells found in this material show a gluing together of the cilia with very little or no propulsive motion. After a course of bronchoscopic drainage, combined with sinus drainage, the cilia of the epithelial cells are free of gluelike secretion.

From the Standpoint of the Roentgenologist

Dr. Willis F. Manges: Dr. Manges considers the relation between sinus disease and lung pathology well established. He does not contend, however, that all chronic sinus infections produce definite lung infections, nor that all chronic lung infections are due to sinus disease. Roentgenology has played an important rôle in the diagnosis and prognosis of these conditions.

Conditions are frequently met in the *x*-ray study of the accessory sinuses that present difficulties in the way of interpretation and are, perhaps, conducive to errors. These difficulties might be considered under three general heads: first, variation in the size of the head and thickness of the bones; second, variation in the shape of the skull and, particularly, the relations of the bones at the base of the skull with the accessory sinuses; third, radiographic exposure technique.

(These subjects were then discussed in detail and the technical points involved thoroughly expounded.)

In the diagnosis of lung changes, roentgenology ranks first in importance. He finds three general types of changes in association with sinus infection: first, is the general peribronchial thickening due, evidently, to a generalized chronic bronchitis; next, there is the type in which the glands at the roots of the lungs and other tissues in this region are markedly enlarged and produce bronchial irritation and, particularly obstruction by pressure; then, there is the more or less localized chronic infection in the lower lobes which leads frequently to more or less extensive degrees of bronchiectasis. Dr. Manges does not recall a true lung abscess that could be attributed definitely to sinus infection as a cause unless there was also operative interference with the sinus infection.

In cases where the lung lesion has reached the stage of bronchiectasis, the sinus disease is so well established that both *x*-ray diagnosis and clinical diagnosis should be made very easily. In cases where the lesion in the lung is well established at the root area, sinus disease is also in a chronic or advanced state of development, and this is probably true also of the cases of well-established general peribronchial thickening. In these the treatment must be directed not only to the sinuses, but also to the lungs.

It is not difficult to determine radiographically that there is evidence of disease in a lung, but it is difficult at times to determine from a single examination whether the lesion is acute or chronic. For example, it is well known that when fibrosis takes place in lung tissue the mediastinal structures are drawn to the side of the fibrosis. This same transfer of the mediastinal structures will occur if portions of the lung become atelectatic, due to bronchial obstruction. In the one case the condition is permanent; in the other the displacement disappears with resumption of function in the lung. Radiographically they may be very difficult to differentiate at the stage when the displacement is most marked. Then, too, the bronchial

shadows are increased during an acute bronchitis and have very much the same appearance as when they are enlarged from a chronic bronchitis, and also exudate in the lower lobe bronchi during an acute stage may give very much the appearance of a bronchiectasis, so that accurate differentiation of the stage of a disease sometimes must be made only after repeated examinations or with the aid of the bronchoscopist, either in getting information by direct vision or by the instillation of iodized oil to determine the presence of bronchiectasis.

These lung changes are particularly recognizable in children during acute stages, and in many instances a diagnosis of tuberculosis has been made from radiographic evidence of enlargement at the root of the lung, when the root glands and peribronchial glands have been infected from antral disease. Dr. Manges presented two cases to illustrate his point. In the first case, the child had not had attention to the sinus disease and it developed into a chronic state and probably will never be entirely cured. With this there have been repeated lung infections. These are now becoming chronic, even though greatly benefited by bronchoscopic treatment. In the other case, the sinus disease was recognized and properly treated, clearing up promptly, and there is hope that eventually a cure will be effected.

Sinus infection occurs in children of early age; it may exist without the presence of nasal discharge or any definite outward sign of infection; it does tend to recur when it does clear up and eventually becomes a chronic process involving many or all of the sinuses. As a result of radiographic study of the sinuses, neck and chest in every patient who has any sign whatever of respiratory disease (for Dr. Chevalier Jackson), it frequently happens that disease of the accessory sinuses is found, although neither the patient nor his physician has known anything about it.

APRIL 16

Some Notes on Agranulocytic Angina; Report of Case With Apparent Recovery

Dr. James A. Babbitt and Dr. Thomas Fitz-Hugh, Jr.: The propriety of considering agranulocytic angina as a separate syndrome is admittedly open to question; but the condition, first clearly recognized by Schultz, in 1922, is sufficiently distinct to warrant,

at least for the present, a specific designation. In analyzing the hundred or more cases in the literature, there is a fairly uniform picture of necrotizing pharyngitis, glossitis, stomatitis and sometimes vaginitis and proctitis, with fever and marked granulocytopenia, terminating fatally in about 90 per cent of the recorded cases. More than 80 per cent have been women. Age variation has been from four and a half to sixty-five years.

The marked ulcerative and necrotic disturbance in the oropharyngeal field suggests a local portal of entry, but the manifold organisms obtained by smear and the minor rôle of adenopathy contradict this, as does the fact that granulocytopenia may precede the ulcerations in some instances. Vincent's organisms are not found invariably enough to prove their causal connection. A certain number of positive blood cultures have been obtained, but often they are negative, and the multiplicity of organisms obtained suggests the possibility of terminal invasion. The characteristic blood picture would seem to locate the trouble in the bone-marrow. This emphasizes the importance of immediate blood studies in any suggestive lesion. Of great interest is the fact that so many of the attacks have been preceded by certain shock elements, such as bone fractures, dental extraction, administration of neoarsphenamine and typhoid vaccine, arthritis and thyroid removal.

The syndrome of agranulocytic angina is threefold: (*a*) an acute systemic infection with prostrating fever; (*b*) a blood picture with white-cell count markedly lowered and polymorphonuclears greatly diminished or absent—other blood elements, including platelets, relatively normal; (*c*) ulcerative and necrotic lesions of oropharynx, often including lips, gums, tongue, pillars, tonsils, pharynx and sometimes intestinal tract and genital areas.

The patient, a nurse, aged twenty-six years, was admitted to the hospital with a temperature of 104° F. and a pulse-rate of 130. The temperature ranged from 102° to 105° F. for seven days, and did not strike normal until six weeks later. She was semicomatose and delirious at times. Both Wassermann and blood cultures were negative. There had been an attack of Vincent's angina several years ago with recovery. A year prior to the present illness the patient had been rather exsanguinated by a ruptured ovarian cyst, requiring transfusion and emergency operation, her leukocytes at that time responding with the usual polymorphonuclear leukocytosis. The present attack immediately followed the extraction of

a left upper molar tooth, with very little bleeding, under gas anesthesia. The following day the patient became increasingly weak and two days later ulcers appeared in the mouth. Mediastinal soreness and a mild papular eruption on the face were noted. There was no jaundice. Several purpuric areas appeared on the second day, but none thereafter. The patient gave a history of hives following the ingestion of tomatoes and strawberries, but presented no history of asthma in herself or family. Both maxillary sinuses were cloudy by *x*-ray, but the other sinuses were clear.

The treatment consisted in irradiation of the long bones, frequent transfusion and ceaseless vigilance in topical oropharyngeal attention. In addition, liver extract, codliver oil, calcium and large amounts of orange juice were administered. It is impossible to say which of these measures were most effective. The patient is doing active nursing today and presents a normal leukocyte count eight months after recovery.

Discussion. Dr. O. H. Perry Pepper: Dr. Babbitt did not exaggerate in any way the illness of this patient. If there is anything of value in trying to separate the Schultz syndrome from the other leukopenias, it is to point out that an overwhelming infection can produce leukopenia. Four of these cases which I have studied did not have an overwhelming infection and this case was not one of overwhelming infection. The leukopenia probably precedes the other manifestations. There may be lesions in the rectum, vagina, nose, mouth or, as in one case, around the fingers. The lesions may only appear at points most susceptible to the neighboring flora. Many organisms have been reported as being present. Does the leukopenia occur first? I have a male patient who reports to me when his leukocyte count drops. He has had agranulocytic angina with four relapses and has had no lesions whatever. His white cells may swing as low as 3000 and later in the week rise to 8000. Of the 4 cases I am familiar with, all were allergic individuals. Anaphylactic manifestations include leukopenia. Dogs and other animals present this phenomenon during anaphylactic shock. Allergy is present in 10 per cent of the population. Is this syndrome an allergic manifestation? The leukopenia certainly strengthens the suspicion. Dr. Blumer suggests that the bacterium was previously present in the throat and all of his cases had a previous infection in the throat. If this is true, we should be especially careful in the examination of the blood. Local treatment is probably the least important; blood transfusion is the most valuable treatment. However, Minot treated one case by venesection and the patient got well; this patient was treated by blood transfusion and got well. Which is the better method? I hope you will watch these cases from the blood standpoint.

Dr. Robert J. Hunter: I have been intensely interested in Dr. Babbitt's paper and Dr. Pepper's discussion. I asked Dr. Pepper about cases with

injury to the long bones when I wrote my paper. An interesting point about the case I reported was that that patient had epilepsy. Therefore, the epilepsy may have been an allergic type. That case appeared to follow injury to the long bones. I would like to ask Dr. Babbitt what led to the extraction of the tooth.

Dr. Matthew S. Ersner: I believe Dr. Pepper struck the keynote as to etiology. We have been following allergic cases recently. A dermatologist told me recently that almost every skin condition is today regarded as an allergic phenomenon. Just as certain cells, as eosinophils, may be stimulated in allergy and increased in number, in other conditions they may be inhibited. An exogenous condition may produce allergy. In entering the long bones injury may be done the bone-marrow or enough absorption occur to further damage the bone-marrow.

Dr. Babbitt (in closing): In studying this case we came more and more to feel the shadowy boundary of this case in the otolaryngological field. The sloughs and necrotic areas were foul and extensive. I do not feel that this portion of the treatment was the important part. I do not know just why this tooth was extracted.

Bursa Pharyngea. (Lantern Demonstration.)

Dr. George M. Dorrance: Dr. Dorrance first became interested in the bursa pharyngea when he observed it in an adult with a cleft-palate. In this individual there was no tonsillar adenoid tissue in the pharynx, and yet there was a distinct pouch or sac, the opening of which was situated above the upper border of the superior constrictor muscle of the pharynx. This opening could be probed for perhaps ½ inch.

The term designates a saclike depression in the posterior wall of the nasopharynx just above the uppermost fibers of the superior constrictor muscle, and usually extending upward and backward toward the occipital bone, where sometimes it reaches this bone with its apex. The bursa is a well-marked structure in the human embryo. Although it is not constant, appearing before the development of the pharyngeal tonsil, its presence in the embryo is conceded by practically all authorities. It seems to be rare as a distinct structure in the adult, and the appearance of a median depression in the pharyngeal tonsil has been confused with a true bursa, so that the presence of a true bursa in adults has been denied.

Dr. Dorrance does not doubt that in certain adults there is a true bursa which is the direct result of the bursa of the embryo, that it ascends upward and backward toward the occipital bone, and

that it is sometimes present, as in this case, even when adenoid tissue is absent. As a rule, the bursa pharyngea requires no treatment. It originates in the embryo as the result of the contact of the notocord with the posterior aspect of the pharyngeal entoderm at a definite point, namely, just above the level of the uppermost fibers of the superior constrictor muscle of the pharynx. It appears as the notocord comes to occupy a relatively more posterior position in the embryo during its intrauterine life. The notocord or its sheath exert direct traction on the entoderm at the site of the development of the bursa pharyngea. This action of the notocord and its sheath may be assisted, perhaps, by traction exerted on the entoderm by certain bands, fasciæ or ligaments. Perhaps an additional factor in the development of the bursa is a natural tendency for the entoderm to invaginate at the site of development of the bursa. Bursa pharyngea has no relationship to cleft-palate, and, therefore, is an independent entity.

Discussion. Dr. George B. Wood: It is scarcely possible to add more after this very complete study of Dr. Dorrance's. This structure has been studied by anatomists and physiologists. It has been definitely shown that it has nothing to do with the canal going up to the hypophysis. It appears just before the pharyngeal tonsil appears in the embryo. At that time it is a mere depression. At the bottom of this groove is the deposit which later goes on to form the pharyngeal tonsil. The faucial tonsil forms by differentiation of cells in the mesenchyme. I have a few slides of tonsil formation in the embryos of hogs. I obtained these embryos from abattoirs in Philadelphia. The epithelium shows marked penetration later into the mesenchyme, but the first evidence of tonsil tissue in the hog is a break in the basement membrane and the basal cells grow irregularly into the mesenchyme. This epithelium that forms tonsils grows in just as the pharyngeal tonsil does and here there is no notocord. It keeps on growing and forms a pocket almost as big as the bursa pharyngeus. I fail to see any better explanation for bursa pharyngea than that it is the anlage of the pharyngeal tonsil. The pharyngeal tonsil is a definite accumulation of lymphoid tissue, and wherever we have an accumulation of lymphoid tissue we find slight penetration of epithelium in that place. I want to correct a term erroneously applied to this body. A bursa is an enclosed sac, lined with synovial membrane, placed where it will reduce friction during movement. This is not a bursa. The slides Dr. Dorrance has presented might be used just as advantageously to prove this body to be the anlage of the pharyngeal tonsil.

Dr. Dorrance (in closing): I think the name is a bad one, but found it attached to it. I did not feel like changing it.

Regeneration of the Mucous Membrane of the Frontal Sinuses

Dr. George M. Coates and Dr. Matthew S. Ersner: Three separate operations were performed upon a dog and satisfactory recovery followed each "radical" operation on the frontal sinuses. The first operation consisted in opening both frontal sinuses externally, the external plate being taken away and the mucous membrane removed as nearly intact as possible for study and for comparison with the replacing membrane to be removed at a later operation. The nasofrontal duct was curetted on the right side, but upon the left the skin and periosteum were united with silk sutures. Healing was rapid and uneventful.

Two months and ten days after the first operation the right frontal sinus was investigated. The outer table had partially reformed with new bone, and the sinus was lined with membrane thicker than normal. The entire anterior wall and the lining membrane were again removed and the wound closed. The third operation, seven months later, revealed the right frontal sinus to be again filled with thick mucous membrane and the anterior wall had regenerated. The membrane was thinner and more normal in appearance than at the second operation, probably due to the greater lapse of time. The left frontal sinus was opened for the first time since its original entry, an interval of nine months. The mucous membrane was found to be thin and delicate and to resemble normal tissue.

The normal mucous membrane showed no glandular tissue. It was thin and somewhat convoluted. The supporting stroma is a delicate fibrillar tissue containing a few spindle-shaped nuclei and a few mononuclear leukocytes. Several small capillaries were observed. A single layer of columnar epithelium covers the surface, which forms folds or papilla-like projections. The specimen taken from the right frontal sinus at the second operation resembled the normal excepting for a few more mononuclear leukocytes in the supporting stroma. The specimen taken from the right frontal sinus at the third operation showed decidedly more connective tissue. The supporting stroma contained a greater number of fibroblasts, and in some places there was edema. Many more mononuclear leukocytes were present and there was a tendency to focal arrangement. Subsurface acini were recognized and the lining cells were hydropic. The surface epithelium was mostly of the stratified columnar variety.

The specimen taken from the left frontal sinus after nine months resembled the final specimen taken from the left frontal sinus excepting for more evidence of infection (hyperemia, hemorrhage, edema and polymorphonuclear leukocytes).

These experiments produced results similar to those obtained by Knowlton and McGregor, excepting that neither goblet cells nor glands were noted in the normal sinus lining. Glandular tissue did appear in the later regenerated epithelial lining, where was also observed the presence of inflammatory elements. Regenerated mucous membrane will again reform after being removed, although the resulting epithelium cannot be classified as entirely normal.

Discussion. Dr. Ersner: The findings have clinical significance. The dog was operated upon several times, and after eight weeks regenerated mucous membrane much like the normal was found. Later, the tissues were found to be thickened, due to infection, and the mononuclear cells predominated, with fibrosis present. At the end of nine months acute infection was found to be taking place in a new mucous membrane. Here we find a condition applicable to the asthmatic. Such individuals may be "cured" after operation and suffer a recurrence after a year or two. If recurrence occurs in animals it occurs in man. We must be kind to other operators.

Dr. Coates (in closing): We were disappointed in not finding glands in the mucous membrane removed at the first operation. This may be due to faulty technique. We did find glandular elements at the end of the second and third operations.

NOVEMBER 19

Presentation of a New Type of Tracheotomy Tube With Valve

Dr. Gabriel Tucker: Most tubes devised for the treatment of laryngeal stenosis possess valve arrangements which occupy too much space. The ideal tube for emergency tracheotomy is the Jackson tube, because it is sufficiently long to reach the trachea. After the wound has healed we face a different proposition; the Jackson cannula now touches the posterior wall, as shown in roentgenograms. The Luer type cannula is found to strike the anterior wall of the trachea. This new tube is intermediate in length between the Jackson and the Luer tubes, is fitted with a valve which functions in a No. 4 tube and serves its purpose better than the older types now in use.

The Present Status of the Intracarotid Method of Treatment of Pneumococcus and Streptococcus Meningitis

Dr. John A. Kolmer: Pneumococcus and streptococcus meningitis is still rare in ear and sinus disease, yet the mortality is so high that this factor is impressive. Experimental work on dogs and cats demonstrated that by "quadruple treatment" survival of 30 per cent of the infected animals is possible. By continued efforts an intracarotid method of injecting serum and drugs in the common carotid arteries was developed in the assumption that such a method would bring therapeutic agents into immediate contact with the meninges; 40 to 60 per cent of animals with experimentally produced meningitis recovered with the new treatment.

In man the technique was employed in 7 cases of hemolytic streptococcus meningitis and 4 cases of pneumococcus meningitis. Not much was accomplished with the streptococcic infection because of the lack of a suitable antistreptococcic serum. One case of pneumococcic meningitis, secondary to an ethmoiditis, recovered. Here the pneumococcus proved to be Type I. A small child and 2 adults with well-advanced meningitis, very cloudy spinal fluid and several thousand cells per cubic centimeter in the spinal fluid died. Of these 1 was moribund when the intracarotid injection was started. The successful patient was given the antistreptococcus serum of Felton, which contains a large amount of Type I, a small amount of Type II, a trace of Type III and no Type IV antibodies. Felton's serum is dispensed in syringes containing 10 to 12 cc. (at least 10,000 units of antibody for Type I pneumococcus).

The serum is injected into each common carotid artery, exposed surgically, using a No. 22 needle and a syringe. There is practically no hemorrhage and no ligature is required. Ten to 12 cc. of serum is injected into each carotid and merely a guide ligature left under each artery and guide clamps left in place because two or three injections daily may be needed. One cubic centimeter of a 1 to 200 solution of optochin hydrochlorid may be added to each syringe.

Four cases with 1 survival is a disappointingly low percentage, but if this technique could be applied as early to human cases as to experimental animals the results should be equally good. Hesitancy to resort to such radical methods and the resultant delay are fatal. Emphasis must be laid on early diagnosis. As soon as

pneumococci are found in the spinal fluid the more radical plan of treatment should be tried immediately, regardless of any other finding, otherwise the patient will die anyway. Ordinary intraspinal treatment is a failure.

During the last year streptococcic meningitis has been more common than pneumococcic meningitis. Reliable scarlet fever antitoxin is useful in treating streptococcic meningitis and is better than the polyvalent sera. Most infections from the mastoid are hemolytic and extremely toxic. Ordinary polyvalent sera, Pragel's iodin, gentian violet, metaphen and neutral acriflavin are as safely given by intracarotid injection as intravenously or intraspinally. In addition we get immediate contact with the meninges. After introducing 10 to 12 cc. of scarlet fever antitoxin in each carotid there is almost an immediate improvement. The spinal fluid shows a sharp reduction of pus cells per cubic centimeter, and on three occasions it was thought recovery would occur. One case perished of frontal lobe abscess later on. Further developments must await the preparation of more potent sera. As far as chemotherapy is concerned, we are only touching the surface and we need more powerful agents. Intracarotid administration marks a distinct advance in meningitis medication. The method is reported to have saved several cases of meningococcic meningitis resistant to intraspinal medication.

The Specific Prophylaxis of Pneumococcus and Streptococcus Meningitis

Dr. John A. Kolmer and Dr. Amano (by invitation): It was found that animals injected in the cisterna magna with the organisms under discussion gave evidence of meningitis in ten to fifteen hours and died in two or three days. Cultures introduced into the tympanic cavities of monkeys produced otitis media, but in no instance meningitis. When Type I Felton's pneumococcic serum (0.5 cc. per kilo of body weight) was also administered the onset of meningitis was delayed and death did not occur for five days and fifteen hours. The control animal died in twenty hours. It was found that 0.2 cc. of serum with 30 mg. optochin hydrochlorid per kilogram of body weight, when administered at the time of the infection, enabled the animals to survive three or four days, while the controls died in thirteen hours to one day, twelve hours.

Most of the experiments were performed with rabbits because the introduction of cultures in the tympanic cavities of rabbits was usually followed by meningitis. Untreated controls died in two to five days. When 0.2 cc. serum per kilo was given intravenously the majority survived indefinitely. The serum was given immediately after streptococci had been placed in the tympanic cavity. When serum was given intramuscularly at the same time the cavity was infected, 1 died in two days and 3 others survived; 0.4 cc. of serum per kilogram of body weight enabled the animals to invariably survive.

It seems possible that in man with Type I pneumococcic infections derived from the ethmoidal or sphenoidal sinuses or the mastoid area, early administration of Type I serum might prevent involvement of the meninges. When optochin hydrochlorid (30 mg.) and serum (0.2 cc.) were given intramuscularly all the animals survived. There was a complete series of survivals when the medication was given by vein. It was evident that in Type I pneumococcic infections Felton's serum, in 0.2- to 1-cc. doses per kilogram of body weight, would offset meningitis in animals when given intramuscularly or intravenously. Whether the addition of optochin to the serum is of advantage is not clear. Given by mouth, some prophylactic action is noted, but the serum is the more effective agent of the two. Optochin is not very effective alone.

Of the untreated controls with Type II pneumococcic infections, 83 per cent died (average of one day, six hours). A similar group treated with Felton's serum had a mortality of 34 per cent. This would indicate that the treatment of Type II pneumococcic cases requires at least twice the dosage used in Type I infections. There is so little antibody against Type III pneumococcus that 63 per cent of the treated cases died, while 66 per cent of the untreated cases died.

It is difficult to produce streptococcic meningitis in dogs and rabbits. These experiments on prophylaxis required the introduction of cultures into the cisterna magna. Untreated animals died in an average of thirteen hours to one day, while those receiving scarlet fever antitoxin lived from two days, thirteen hours to five days, three hours. From the standpoint of recovery, the percentage of success is not as impressive as in pneumococcus infections.

Streptococcic meningitis remains more lethal than the pneumococcic form because therapeusis is less effective. Bacteriological

studies must be made very early. If pneumococci are found and meningitis is suspected clinically, it is recommended that Felton's concentrated antipneumococcus serum be given intravenously (10 cc. for an adult, 5 cc. for a child). Later the same dose may be given intramuscularly, repeating the dose on the third day. Enough antibody may gain access to the meninges to prevent meningitis. Immediate bacteriological examinations are essential, so that if pneumococcus Type I or II are found something worth while may be done to prevent meningitis. If hemolytic streptococci are found one of the scarlet fever antitoxins should be used. The latter may produce reactions while the pneumococcus sera produce no reaction.

Discussion. Dr. George B. Wood: I have not seen much meningitis, but we all appreciate the work Dr. Kolmer is doing to help us combat these disastrous infections. The prophylactic viewpoint is instructive. We cannot give serum to every case of streptococcic otitis media, but when the pneumococcus is present the matter is different. A case of pneumococcic otitis media, with profuse discharge and a positive culture reported in twelve hours, was given Huntoon's antibodies and the pneumococci had disappeared from the ear in two days. Huntoon's antibodies do not give so severe a reaction that they cannot be given in otitis media, and I believe such measures should be used in treating otitis media.

Dr. Amano: The tympanic cavity of the dog is so deeply placed that a surgical approach is necessary to infect it. In some instances the tympanic membrane may be curetted. In one series following tympanic application the untreated animals showed a mortality of 77 per cent, while the treated animals had a mortality of 13 per cent.

Dr. Francis C. Grant: It is a pity that we do not have more researches of this type, well thought out at the beginning and carefully controlled. I wish to ask if some of my impressions are correct. The protective mechanisms of the body in combating infection produce adhesions of the meninges, and in this event serum cannot be brought to the blocked-off area by the intraspinal route. The only route of approach is intracarotid. I have seen many mastoid cases which looked as if they might be meningitis for twenty-four hours and then all blew over and nothing developed. We should use these methods early, but, since we often see children with flare-ups which later prove to be meningismus and not meningitis, there is some hesitancy in using serum. I have never seen a case of pneumococcic meningitis recover. We usually hope the symptoms reflect a serous meningitis or meningismus and thus therapy may not be required. This therapy to be effective must be used early. I have seen cases of pneumococcic meningitis do well for a time and then relapse. Some mastoid cases go into meningitis because the mastoid area has not been cleaned up. Such cases might have the primary meningitis controlled by Dr. Kolmer's technique, and then have a second flare-up from the incompletely cleaned

up mastoid area and another meningitis. Sperling reported 8 cases with 5 recoveries, due to laminectomy and permanent drainage through the laminectomy wound, with the administration of large quantities of water. We tried the same treatment, but our cases all died. I would like to ask Dr. Kolmer if it is possible to inject the semicircular canals rather than the tympanic cavity?

Dr. Amano: Labyrinthinitis is one of the first steps to meningeal complications. We do not know whether we caused labyrinthinitis first or if hematogenous spread occurred. Some of our animals were injected into the labyrinth. In dogs we opened the cochlea. In rabbits pneumonia followed such procedures, so that we had to pass a needle through the overlying muscular structures and so penetrate the middle ear. The simplest method is administration through the drum. I am sure many of these animals developed labyrinthinitis.

Dr. E. B. Gleason: A child may show symptoms of meningitis and these may disappear after the mastoid operation. When meningitis appears after a mastoid operation it is likely that the meningitis will persist and develop.

Dr. Amano: The frequency of meningeal infections secondary to infection in the mastoid area is supposed to be about 1 in 300 or 1 in 150.

Dr. George M. Coates: In answering Dr. Grant I would like to say that we do not see many cases of meningitis after mastoid operations. There may be a cloudy spinal fluid with 2000 cells per c.mm., but no organisms; but the case clears up after operation and we assume it was a serous meningitis. Some do develop, however, and we cannot tell whether profuse suppuration will occur or whether the process will subside. In a study of 650 mastoid cases there was found a mortality of 3.5 to 4 per cent and since many of these were late cases there was apparently not so much meningitis. How many of us leave infection in the mastoid? We are cleaning the mastoids much more thoroughly than we did twenty years ago; but, like dealing with ethmoidal cells, it is frequently hard to get every cell.

Dr. Ralph Butler: Most meningitis occurs before operation for mastoid. Dr. Kolmer has given us a very good idea as to prophylactic measures. If we could use this method early in otitis media it should prove effectual. Several cases of meningitis occurring before operation were given intracarotid injections several years ago, but all died. Our sera may be more potent now.

Dr. Kolmer (in closing): While the otolaryngologist is now able to leave a clean field after mastoid procedure, it is much more difficult to thoroughly remove all infected cells in ethmoidal infections. I have seen many ethmoidal cases where the surgeon had to be "jacked up" about residual infection. Dr. Gleason emphasizes the difficulty of diagnosis, and this is a point well taken. I know at least two cases of pneumococcus meningitis which recovered, but the spinal fluid was clear in both instances and the pneumococci were gotten only in culture. This is not the type I mean. I mean those with cloudy spinal fluid and with abundant organisms in the fluid. What are we going to do with a strongly positive globulin reaction

and 1000 cells per c.mm. in the spinal fluid? Shall we wait or use this therapy? I think it is safer to operate and use intracarotid therapy because there is no danger to the patient. All surgeons doing otolaryngology are capable of using the carotid for this purpose.

Treatment of Stricture of the Oral Pharynx.

Dr. George M. Dorrance: Stricture of the oral pharynx results from the swallowing of acids or caustics, and occasionally occurs as a complication of ulcerative syphilis of the pharynx and on rare occasions follows tonsillectomy. Congenital strictures at this location are unreported. Treatment by gradual or rapid dilatation of the opening is unsatisfactory as the scar tissue invariably contracts again.

The subject of this contribution had a stricture of the oral pharynx following tonsillectomy. The opening in the pharynx was situated on the posterior surface of the tongue and the edges of the stricture were rigid. Tracheotomy was done and the opening permitted to close. An incision was made on either side of the stricture and a cavity made with a hemostat through the lateral wounds. The cavities were enlarged upward and specially constructed vulcanite pegs introduced, first pushing them well below the stricture and then pressing them upward into the preformed cavity to prevent each peg from jumping out. The vulcanite capsules were then removed and washed. Two large Thiersch skin-grafts were then cut and placed over the vulcanite capsules with the skin surfaces toward the capsule. The capsules were then reintroduced into the cavities and the wounds closed by sutures. The capsules were in place two weeks when an incision was made on either side of the stricture down to and through the skin-grafts and the capsules removed. A skin-lined cavity remained on either side of the previously existing stricture. By pressure these cavities were converted into a single cavity. A small rubber-ring pessary was then introduced and allowed to remain two weeks. The pessary was removed for purposes of cleansing every few days. The patient is now able to swallow a normal mixed diet and there is no respiratory difficulty. It has been approximately two and a half years since the operation.

Discussion. Dr. Fielding O. Lewis: I have nothing to add. This patient had had all sorts of treatment. Electrocoagulation and fulguration had been tried without success. I can vouch for all Dr. Dorrance has said about the recovery.

Dr. George M. Coates: Does the presence of the skin-grafts affect swallowing? How does skin act in the moist medium of this region?

Dr. Dorrance: This tissue resembles mucous membrane now, and under the microscope looks like neither mucous membrane nor skin. It is moist, just like mucous membrane. I have not been able to try this technique on a caustic contracture.

DECEMBER 17

Orbital Abscess Following Sinus Disease

John R. Davies, Jr.: The patient in question, a girl, aged fourteen years, had had her tonsils and adenoid tissue removed at the age of two years. She was not especially subject to head colds, but when they occurred tended to be prolonged. When first examined, a head cold had been present for ten days and, dating from the preceding day, there had been considerable edema of the right eyelid, some protrusion of the eyeball and a slight edema of the conjunctiva. The patient was decidedly toxic and had much pain over the right antrum. The mucosa in the right nostril was swollen and the nostril was filled with pus. The temperature was 101° F.

The roentgenological report from wet films indicated pus in the right antrum, but the right frontal and ethmoidal sinuses were indefinite. The leukocyte count was 6200 and the urine was loaded with albumin and erythrocytes. Because of the age and sex of the patient the frontal sinus was not opened in view of the vague roentgenological findings. A Caldwell-Luc operation revealed considerable pus and many polypi in the antrum. The Staphylococcus aureus hemolyticus was recovered from the pus in pure culture. The following morning there was increased swelling of the right eyelid, marked edema of the conjunctiva and protuberance of the eyeball. The right frontal sinus was opened through the usual incision in the eyebrow and considerable pus evacuated. The roof of the orbit was opened and free pus found. Very little pus was found in the ethmoidal cells.

The incision in the eyebrow was closed with dermal sutures and drainage maintained intranasally. Twenty-seven days after admission to the hospital the patient was discharged with ocular move-

ments and vision of the right eye normal. A year after operation the sinuses are clear upon transillumination and the eye is normal.

The case illustrates difficulties frequently met in the early diagnosis of sinus disease, in the absence of headache or pain over the sinus on pressure. In the presence of repeated or prolonged head colds, with vague roentgenological reports, indefinite findings in the nose and upon transillumination, is the exploratory puncture of the antrum a safe and logical procedure, and might it have prevented this complication?

Discussion. Dr. J. Thorington: This case was the most remarkable of its type that I have ever encountered. The conjunctiva was protruding between the lids, the retinal vessels were tortuous and full, but, fortunately, no break had occurred in the vessels. It was interesting to observe how rapidly the edema disappeared. There had been proptosis of ½ inch on the affected side and some on the opposite side.

Dr. Fielding O. Lewis: In this connection I would like to mention the case of an infant, aged fifteen days, with proptosis due to disease of the ethmoidal sinuses. The diagnosis was corroborated by roentgenological and operative findings, but the patient died.

Dr. George Fetterolf: A somewhat similar case at the University Hospital, with some edema of the tissues about the eye on the uninvolved side, was found at operation to have an opening in the interfrontal septum. I was curious as to whether such an opening in the interfrontal septum was found in this case at operation.

Dr. Davies: The interfrontal septum was found to be intact at operation.

Acute Mastoiditis With Basilar Meningitis

Dr. William Hewson: The patient, a male, aged forty-one years, developed a right acute otitis media about nine weeks after an acute cold. Incision was promptly made and, after draining for four weeks, a furuncle developed in the right external auditory canal, followed by a postauricular abscess. Drainage of these abscesses continued for two weeks, accompanied by a persistent right-sided headache and intermittent nausea. There was no vertigo. At this time the patient was admitted to the hospital with a temperature of 100.6° F; pulse, 94; respirations, 22. The pupils reacted to light and accommodation, and there was no impairment of ocular movements. There was a little tenderness over the mastoid area and the external canal was a trifle edematous. The drum membrane was inflamed and slightly bulbing, and was reincised. The nasal septum was deflected in its midportion, crowding the middle

turbinate on the right. Mucopus was visible in the middle and inferior meati on the right. The pharynx was congested, as well as a small residual tab of the right tonsil.

A roentgenogram revealed uneven cloudiness throughout the right mastoid area and localized areas of absorption in the antrum in the region of the knee of the sinus toward the tip, the appearance suggesting acute suppurative mastoiditis. From the second to the eighth day in the hospital there was improvement. On the eighth day nausea and vomiting appeared, with a chill and temperature of 102° F. The following day there was stiffness of the neck and increased reflex response. The leukocytes were 24,000, with 95 per cent polymorphonuclears. The spinal fluid, under a pressure of 40 mm., was cloudy and contained 12,000 leukocytes.

At operation the mastoid cortex was firm and thick. A fair-sized abscess cavity occupied the tip of the mastoid, but the lateral sinus was found to be normal. In following the zygomatic cells a necrosed area was reached which communicated by a fistulous tract through the additus ad antrum. The dura over this area was inflamed and adherent, and a small perforation was found in the dura opposite the fistulous tract. The wound was left open and packed. The following day the temperature rose to 105° F. and the patient died. Pneumococci were cultured from the spinal fluid, and at autopsy a localized pachymeningitis was found to involve the pons and medulla, especially on the right. An area of necrosis involved the petrous portion of the temporal bone.

This case illustrates the possibility of an acute rhinitis developing into an ethmoiditis, with extension up the right Eustachian tube to the middle-ear to produce purulent otitis media. Cells in the immediate vicinity of the aditus were probably first attacked and extension involved the neighboring cells in the petrous pyramid. The breaking down of cells in the posterior root of the zygoma made the fistulous tract which extended through the periosteum at this point. Extension is thus possible from mastoid cells to the cells in the petrous pyramid.

Discussion. Dr. Louis Baer: I recently had a case similar to the one reported by Dr. Hewson. I would like to know when unconsciousness came on. My case was due to the streptococcus. The drain was taken from the wound on the ninth day. On the eleventh day there was headache and a temperature of 99° F. The drain was reinserted and taken out two days later. For five days there was a temperature of about 99° F. or

99.5° F. At consultation it was thought there might be some retention. The following morning the patient could not be aroused. The streptococcic type is more virulent than the pneumococcic type and is less likely to recover. Serums, vaccine, bacteriophage and other measures should be used early to prevent extension of the infection.

Dr. B. Alexander Randall: Pachymeningitis is a slight affair, but with drainage usually recovers. It is only with penetration of the arachnoid structures, with arachnoid involvement, that there is danger. In pachymeningitis there is thickening of the membrane and granulations form on the outer surface, which are more impervious to infection than the usual membrane. Frequently actual penetration and opening up of the arachnoid cavity occur, as in this case, with pus in the spinal fluid, and only vigorous treatment will avail. I believe much can be done by washing out these cavities, even using a spatula, which will push the brain tissue aside. I have washed out in this fashion large quantities of thick pus. It did not always save the patient, but much may be accomplished in this way.

Dr. Fielding O. Lewis: I would like to ask Dr. Hewson if the cultures were from the mastoid or the cerebrospinal fluid.

Dr. Hewson: The culture was from the cerebrospinal fluid, but the same organism was obtained from the mastoid. The patient was conscious almost until death. Dr. Shuster saw this case with me and I would be glad to hear from him.

Dr. B. H. Shuster: The first sign of meningitis was the development of chill. Nausea preceded the other signs of severe illness. Undoubtedly this patient had pachymeningitis early, but at that time no other symptoms of meningitis could be elicited. The furuncle did not clear up soon after incision, leading to the suspicion that bone necrosis might be responsible for its persistence. Many such cases get well with the early symptoms this man had.

Dr. Fielding O. Lewis: It occurred to me that this would have been a suitable one in which to employ the Kolmer treatment.

Compound Fracture of the Malar Bone With Involvement of the Maxillary Sinus.

Dr. Thomas G. Aiken: The patient, who was present, had received a violent blow over the right malar bone, resulting in a compound fracture which later became infected. For six weeks the temperature remained elevated and then fell but never entirely reached normal. At this time Dr. Aiken first saw the case and a roentgenogram revealed the extent of the fracture and indicated increased cloudiness of the antrum. A needle introduced into the antrum released pus and after a series of irrigations the temperature returned to normal. Later the patient returned with a swollen

face and toothache. A dentist extracted one molar tooth having two molar roots and a third root surrounded by a large granuloma. No direct communication of the tooth socket with the antrum could be demonstrated. Two weeks after the extraction pus had almost disappeared, and at the present time there is no pus in the antrum and the sinus is much clearer by transillumination.

Discussion. Dr. George B. Wood: We may expect almost anything from traumatic infection of sinuses. One case admitted to the Graduate Hospital, a woman who had been struck three times by a hammer in the hands of her husband, was unconscious on admission. One blow had struck the frontal area, one the maxillary sinus and the third the mastoid area. The mastoid area was wide open, the frontal sinus was open and the eye had dropped out so that the front walls of the sphenoidal sinuses could be seen through the smashed walls of the ethmoidal cells. When the case was dressed a tremendous cerebrospinal leak was found. Dr. Grant made an osteoplastic flap and lifted the brain from the cribriform plate after turning the dura back from the frontal lobe. The leak could not be found, so the wound was closed and the woman later made a complete recovery.

Dr. Fielding O. Lewis: I saw a woman with a fractured outer wall of the maxillary sinus due to an automobile accident. A very foul and extensive infection followed, which later required a radical operation for relief.

Chondromata of the Larynx. Report of a Case

Dr. Robert M. Lukens: Seventy-one cases of chondromata of the larynx are reported in the literature. They most frequently appear in the cricoid or the thyroid cartilage and usually are sessile and single. They show a tendency to invade adjacent cartilages, but rarely, if ever, metastasize. Masses of new tissue appear, composed of hyaline, elastic or white fibrocartilage, developing from preëxisting cartilage. The tumor is usually rounded, smooth, tuberous or lobulated, with well-marked capsule. Occurrence is rare after puberty, according to many, but the cases reported occurred from the age of fifteen to sixty-six years, so that chondromata of the larynx are tumors of rather late adult life. Even with complete shelling out of the tumor, recurrence is apt to occur, and laryngectomy is required more often than is supposed.

A white male, aged fifty-seven years, was rejected by an industrial medical examiner because of husky voice and the presence of a tumorlike mass in the right side of the neck. The hoarseness was of two years' duration, but there had been no cough, pain, dyspnea

or dysphagia. To the right of the midline of the neck, at the level of and above the cricoid cartilage, was a fairly smooth, hard, bone-like, hemispherical mass, about $\frac{7}{8}$ inch in diameter and about $\frac{1}{2}$ inch thick, firmly attached to the anterior arch of the cricoid and lower portion of the right thyroid ala. Mirror laryngoscopy revealed a marked prominence of the subglottic tissues on the right side. There was no noticeable impairment of motion of the overlying cord except lack of tension of both cords on phonation. No ulcerations were present.

The tumor mass in the neck was removed by external operation under local anesthesia. The patient made a good recovery, but some time later developed attacks of dyspnea with inspiratory, crowing sounds. Three years after the removal of the tumor the laryngeal lumen was greatly narrowed by a rounded mass, which projected inward from the right side, obliterating the anterior portion of the chin, raising the right vocal cord and pushing it inward so that the right ventricle could be inspected readily. The arytenoid was not involved. The subglottic tissues on the left were not involved.

Laryngectomy was indicated, but postponed because of a severe mouth infection. Tracheotomy was necessary, because of the extreme dyspnea. Later, laryngectomy was performed, and the tissue found to be chondroma. A good recovery was made, but the patient became unmanageable later and died of bronchopneumonia eight days after the operation.

PROCEEDINGS

OF THE

SECTION ON GENERAL MEDICINE

PAPERS READ DURING 1930

JANUARY 27

Dr. Joseph V. Klauder, "Some Interesting Case Records of Syphilis."

Dr. Alexander Margolies (by invitation), "Factors Influencing the Intensity of the First Heart Sound."

Dr. Martin E. Rehfuss, "Gastric Digestion of Milk."

Dr. Charles C. Wolferth, "The Clinical Value of Heart Sounds Registration."

Dr. Leon Jonas, "The Choice of Carbohydrate Foods in Diabetes."

Dr. Harold L. Goldburgh (by invitation) and Dr. Henry K. Mohler, "Report of a Case of Diabetes Mellitus Which Required Unusually Large Doses of Insulin."

FEBRUARY 24

Dr. Malcolm Harkins and Dr. Sigmund S. Greenbaum (by invitation), "The Experimental Aspects of Staphylococcus Filtrates."

Dr. Burgess Gordon and Dr. W. M. Cashman (by invitation), "The Incidence of Acquired Tuberculosis in Workers During Service in a Tuberculosis Sanatorium."

Dr. Francis Wood (by invitation) and Dr. Charles C. Wolferth, "Studies on Angina Pectoris, Clinical and Laboratory."

Dr. Frederick J. Kalteyer, "The Relation of Muscular Rigidity to Pain in Abdominal Disease."

Dr. Reynold S. Griffith (by invitation), "A Case of Erythremia with Erythromelalgia."

MARCH 24

Dr. Edward L. Bortz (by invitation), "The Treatment of Obesity."

Dr. Edward Weiss and Dr. Thomas Aceto (by invitation), "Fluoroscopic Visualization of the Introduction of Lipiodol Into the Bronchi by the Passive Technic."

Dr. A. Cantarow (by invitation), "The Tissue Factor in Diabetes Insipidus."

Dr. Armand J. Quick (by invitation), "An Advanced Case of Osteitis Fibrosa. Hyperparathyroidism."

Dr. Thomas M. McMillan and Dr. Samuel Bellet (by invitation), "Treatment of Auricular Flutter."

APRIL 28

Drs. Harry Shay, Eugene Schloss, Isadore Rodis and Milton Bell (all by invitation), "The Metabolism of Galactose. Its Use in Testing Liver Function."

Dr. James E. Cottrell, "Aortic Aneurysm Producing Massive Atelectasis: Case Report."

Dr. Leon Jonas and Dr. Simon S. Leopold, "Idiopathic Tetany."

Dr. David Riesman and Dr. Harold S. Davidson (by invitation), "Psittacosis, with the Report of a Case."

Dr. William E. Robertson and Dr. Samuel Bellet (by invitation), "The Electrocardiographic Evidence of the Action of Digitalis."

MAY 26

Dr. Francis C. Wood (by invitation) and Dr. James E. Cottrell (by invitation), "The Effects of Adrenalin on Angina Pectoris."

Dr. Louis Tuft (by invitation), "Active Immunization Against Typhoid Fever, with Particular Reference to an Intradermal Method."

Dr. Virgil Moon, "Experiments Bearing on the Etiology of Rheumatic Fever."

Dr. Elmer H. Funk and Dr. F. M. Huntoon (by invitation), "Biochemical Studies of Bacterial Derivatives" (XI. Skin Reactions in Man with Human Tubercle Bacillus Protein M. A. 100. Preliminary Report.)

Dr. John Q. Griffith (by invitation), "Capillary Microscopy and Its Clinical Applications."

Dr. John H. Arnett and Dr. Charles Francis Long (by invitation), "A Case of Congenital Pulmonary Stenosis, with Late Onset of Cyanosis and Death from Pancreatic Carcinoma."

OCTOBER 27

Dr. Samuel L. Immerman (by invitation), "The 'Gastric' and Abdominal Symptoms of Irritable Colon: With a Report of Gastric Crisis."

Dr. D. N. Kremer (by invitation), Obesity: "Observations on Treatment by Dietary Measures."

Dr. Jay Frank Schamberg and Dr. H. Brown (by invitation), "Studies in Calcium Metabolism Based upon Animal Experimentation; the Introduction of a New Therapeutic Calcium Compound in Arsphenamine Dermatitis."

Dr. Charles C. Wolferth and Alexander Margolies (by invitation), "The Early Diastolic Snap, a Valuable Auscultation Sign of Mitral Stenosis."

Dr. Thomas Fitz-Hugh, Jr., "Liver Therapy, Its Chemical, Hematologic and Physiologic Effect."

Dr. Joseph V. Klauder, "Laboratory Studies of the Humoral Aspects of Immunity in Syphilis."

NOVEMBER 24

Dr. David Kramer (by invitation), "Tuberculous Meningitis with Syphilitic Meningitis Terminating in Recovery: A Review of the Literature."

Drs. Joseph B. Wolffe, Donald Findlay and Edward Dessen (all by invitation), "Treatment of Angina Pectoris with a Vasodilator Substance."

Dr. Clifford B. Farr, "Routine Sedimentation Tests in Mental and Nervous Cases."

Dr. S. S. Greenbaum (by invitation), "Experimental Study on the Curative Value of Certain Bismuth Compounds in Experimental Syphilis."

DECEMBER 22

Dr. Karl Kornblum (by invitation) and Richard T. Ellison (by invitation), "The Roentgen Diagnosis of Tuberculous Atelectatic Cirrhosis of the Lung."

Dr. R. S. Griffith (by invitation), "Coarctation of the Aorta of the Adult Size."

Dr. F. William Sunderman, "The Total Base and Protein Components of the Serum During Lobar Pneumonia."

Dr. Thomas Fitz-Hugh, Jr., "A Case of Quartan Malaria in an Infant Acquired in the Philadelphia Area."

Dr. Charles C. Wolferth and Dr. Alexander Margolies (by invitation), "Differential Diagnosis of 'Extra' Heart Sounds."

PROCEEDINGS

OF THE

SECTION ON PUBLIC HEALTH AND INDUSTRIAL MEDICINE

PAPERS READ DURING 1930

MARCH 28

Miss Dorothy Wiesner, Statistician of the Philadelphia Health Council (by invitation), "Present Hospital Provision for Tuberculosis."

Dr. James M. Anders, "Some Practical Aspects of the Subject of Additional Hospitals for Tuberculosis Patients in Philadelphia."

PROCEEDINGS

OF THE

SECTION ON MEDICAL HISTORY

PAPERS READ DURING 1930

JANUARY 13

Dr. John H. Moore, Bridgeton, N. J. (by invitation), "Sir Henry Holland, Physician, Scientist, Traveller and Man of Letters."

Dr. J. Hendrie Lloyd, "Benjamin Rush and Psychiatry."

Dr. Wilmer Krusen, "The History of the Philadelphia College of Pharmacy."

FEBRUARY 10

Dr. Randolph Winslow, Baltimore (by invitation), "John Archer, M. D., the First Medical Graduate in America."

Dr. Francis R. Packard, "A Medical 'Cause Célébre'—The Webster-Parkman Murder Case."

MARCH 10

Dr. William Harper Davis, New York City (by invitation), "Sir E. Ray Lankester, Biologist."

Dr. Walter Freeman, Washington, D. C. (by invitation), "Lewis Morgan: The Note Books of a Tory Medical Student."

APRIL 14

Dr. Joseph L. Miller, Thomas, West Va. (by invitation), "Silhouettes of Early Philadelphia Physicians and Others."

Dr. Robert L. Pitfield, "The Medical Student Who Flunked but Won the Nobel Prize."

Dr. Burton Chance, "Nicholas Culpeper."

MAY 12

Dr. Arthur D. Hirschfelder, Minneapolis, Minn. (by invitation), "William Withering."

Dorothea Waley Singer, London, England (by invitation), "Alchemy."

Dr. Charles Singer, London, England (by invitation), "Sidelights on Anatomical Teaching at Padua in Harvey's Day."

NOVEMBER 10

Dr. Leopold Vaccaro (by invitation), "Virgil and His Medical Contemporaries."

Dr. Robert L. Pitfield, "Francis Thompson: The Medical Student and His Box of Toys."

DECEMBER 8

Dr. Herman B. Allyn, "Oliver Wendell Holmes, His Wit and Wisdom."

Sir D'Arcy Power, England (by invitation), "On the Royal Gift of Healing."

DECEMBER 11

Dr. G. Elliot Smith, Professor of Anatomy in the University of London, "The Pekin Man."

ANNUAL REPORT OF THE LIBRARY COMMITTEE FOR 1930

Mr. President: In accordance with the ordinances and By-Laws of the College, I herewith submit the following report of the Library Committee for the year 1930:

Total number of volumes in the Library, including the bound volumes and 26,592 unbound "Reports" and "Transactions"	165,150
Number of unbound "Theses" and "Dissertations"	22,682
Number of unbound Pamphlets	194,237
Manuscripts	438

Included in the above are 4461 volumes known as "Reserves," consisting of second copies of some of the more important periodical publications; also 2950 volumes more or less incomplete.

The duplicates, which number 7284 volumes at this date, are not included in the above total.

The following table shows the number of volumes in the various divisions of the Library:

	Bound.	Incomplete and unbound.	Total.
General Library	118,900	2,950	121,850
Lewis Library	13,850	44	13,894
On permanent deposit:			
S. D. Gross Library	3,778	3	3,781
Library of the Obstetrical Society of Philadelphia	217	0	217
Mütter Museum*	190	0	190
			139,932

Received during the year from all sources, 3058 volumes, 10,331 pamphlets and 18,815 numbers of various periodicals.

* The books of the Mütter Museum are not permitted to be removed from the College building.

Divided as follows:

	Volumes.	Pamphlets.	Journals.
General Library	2,185	10,096	18,369
Lewis Library	5		
S. D. Gross Library	11		
By purchase, General Account	518		
In exchange	339	235	446
	3,058	10,331	18,815

Accessions (including 6 volumes of "Reserves"), 2316.

	Volumes.
General Library	2,300
Lewis Library	5
S. D. Gross Library	11
	2,316

Total increase in the number of volumes for the year, 2316.

Photographs received in response to requests sent out during the past year and voluntary gifts, 56. Fellows of the College, 4; other than Fellows, 52.

Total number of portraits listed to date, 22,417.

The individual "Donors" for the year ending November 1, 1930, number 439; this represents 914 distinct presentations. Each gift is duly acknowledged.

The following list shows individual gifts of twenty volumes and over; also the number of volumes presented by the various publishing houses:

	Volumes.
Dr. Lewis J. Burns	31
Dr. Clifford B. Farr	43
Dr. Herbert Fox	32
Dr. J. P. C. Griffith	27
Dr. R. J. Hunter	38
Dr. William W. Keen	28
Dr. E. B. Krumbhaar	34
Dr. George C. Kuesel	32
Dr. Arthur B. Light	32
Dr. George M. Piersol	25
Dr. Charles S. Potts	28
Presbyterian Hospital	22
Dr. George E. deSchweinitz	22
Dr. Samuel S. Stryker	250
Dr. James E. Talley	56
Dr. T. Mellor Tyson	139

From the publishing houses of:

	Volumes.
P. Blakiston's Son & Co.	13
F. A. Davis Company	14
Paul B. Hoeber	2
Lea & Febiger	26
J. B. Lippincott Company	19
C. V. Mosby Company	1
W. B. Saunders Company	45
B. Westermann Company	1

The library is indebted for large gifts of pamphlets and unbound periodicals to the following donors:

Dr. A. P. C. Ashhurst
Dr. C. W. Burr
Dr. Burton Chance
Dr. S. S. Cohen
Dr. Francis X. Dercum
Dr. W. A. N. Dorland
Dr. George M. Dorrance
Dr. Clifford B. Farr
Dr. J. C. Gittings
Dr. J. P. C. Griffith
Dr. Alfred Hand, Jr.
Dr. Hobart A. Hare
Dr. John H. Jopson
Dr. William W. Keen
Dr. Frederick Krauss
Dr. E. B. Krumbhaar
Dr. F. H. Leavitt
Dr. Joseph Leidy
Dr. Edward Martin
Dr. Charles K. Mills
Dr. C. F. Mitchell
Dr. Elliston J. Morris
Dr. Charles A. O'Reilly
Dr. A. C. Sautter
Dr. Paul J. Sartain
Dr. George E. deSchweinitz
Dr. William J. Taylor
Dr. T. Mellor Tyson
U. S. Public Health Service
University of Pennsylvania
Dr. Joseph Walsh
Dr. James C. Wilson

563 new publications were added to the Library during the past year, viz.: United States, 260; Great Britain and Colonies, 69; France, 74; Germany, 138; Italy, 71; all other countries, 11. Of these new publications, 14 were written or edited by Fellows of the College.

13 volumes were presented by the following authors or editors:

Dr. B. Adachi
Dr. James M. Anders
Dr. W. Seaman Bainbridge
Dr. H. G. Byers
Dr. J. Shelton Horsley
Dr. Samuel A. Levinson
Dr. Charles C. Norris
Dr. Albert O. W. D. Pinson
Dr. W. Allen Pusey
Dr. H. D. Rolleston
Dr. H. C. de Souza-Araujo
Dr. James J. Walsh
Dr. B. W. Weinberger

7 volumes were sent by the publishers at the request of the following authors or editors:

Dr. W. A. N. Dorland
Dr. R. Max Goepp
Dr. Hobart A. Hare
Dr. J. C. Keeler
Dr. J. H. Stokes

Summary of the "Funds":

	Volumes purchased.	Cost.
Henrietta Rush Fales Baker Fund	47	$204.47
Luther S. Bent Fund	16	24.03
William T. Carter Fund	16	125.35
Gerardus Clarkson Fund	5	9.43
Francis X. Dercum Fund	21	110.59
Louis A. Duhring Fund	27	242.17
Augustus A. Eshner Fund	50	179.31
John D. Griscom Fund	58	245.53
Richard H. Harte Fund	46	344.99
Charles T. Hunter Fund	3	14.10
William F. Jenks Fund	44	237.58
Oliver A. Judson Fund	5	27.01
William V. and John M. Keating Fund	7	41.93
William W. Keen Fund	19	102.08
Library Endowment Fund	83	635.30
Horace Magee Memorial Fund	76	628.95
J. Ewing Mears Fund	32	218.01
Charles K. Mills Fund	2	9.41
Weir Mitchell Fund	27	229.75
E. E. Montgomery Fund	14	116.69
John H. Musser Fund	5	13.60
Elizabeth K. Newcomet Fund	5	23.11
William F. Norris Fund	27	156.52
Charles A. Oliver Fund	8	42.65
Philadelphia Medical Society Fund	1	16.75
Lewis Rodman Fund	45	146.10
Douglas Stockton Warren Fund	12	49.44
John F. Weightman Fund	5	55.15
J. William White Fund	24	188.97
Caspar Wistar Fund	35	212.82
S. Lewis Ziegler Fund	0	0.00
	765	$4,651.79
From the General Account—by purchase	518	$6,129.38

Special accounts:

	Volumes purchased.	Cost.
Fund for completing files of journals	77	$355.76
Fund for rare and valuable books	49	702.00
S. D. Gross Library Account	8	232.34

	Expended.
Catalogue Endowment Fund—on account of salaries	$46.50
Louis A. Duhring Fund—for general purposes of the Library	12,000.00
Morris Longstreth Fund—on account of salaries	1,110.00
George B. Wood Fund—Library supplies, stationery, etc.	366.83

Number of volumes bound, 1897.

	Visitors.	Fellows.	Total.
Use of Library	10,921	1,674	12,595

The Library has been kept open two evenings each week and on the five minor legal holdays for the same period of time as during the previous year:

Evenings.	Visitors.	Fellows.	Total.
74	1,190	177	1,367

Legal holidays.	Visitors.	Fellows.	Total.
5	217	30	247

These figures are included in the total given for the year.

	1930.	1929.	Decrease.
Number of volumes consulted in the Library	49,244	52,507	3,263

The number of books "consulted in the Library" includes only those supplied on demand. Readers have access to the bound volumes of periodicals and works of reference kept on the shelves in the Reading Room, and the Fellows of the College have access to the Book Stacks. There are, therefore, many volumes consulted of which no accurate record can be kept.

	1930.	1929.	Decrease.
Number of volumes taken out	3,708	4,368	660

Use of Study Rooms: Average number of rooms in use throughout the year, 4.

Number of volumes in these rooms during the year, 1582.

	Works.	Volumes.	Cards written.	Printed cards revised.
Cataloguing	864	889	5,592	3,198

All books added to the shelves during the past year and 489 of the more important pamphlets have been catalogued and shelf-listed; and all bound volumes, including periodicals, transactions and reports, have been accessioned.

Revision of the Catalogue: 9999 cards have been revised, rewritten and filed.

2985 unbound pamphlets and reprints have been subject-headed and arranged alphabetically by subject and by author under the subject.

We have listed November 1, 1930, 1336 current periodical publications, including "Transactions" and "Reports," obtained through the following sources:

	American.	Foreign.
Endowment Funds	115	208
By purchase from General Account	93	254
In exchange	98	128
Editors	197	53
Publishers	76	28
	579	671
Reports not included in the above	61	25
	640	696

The actual number of periodicals received during the year, including new subscriptions (6 American, 2 Foreign), total 1250.

The following is a list of the foreign schools of medicine with which we exchange publications at the present time:

University of Amsterdam
" Basel
" Berlin
" Bern
" Bonn
" Breslau
" Erlangen
" Giessen
" Göttingen
" Halle
" Heidelberg
" Kiel
University of Königsberg
" Lausanne
" Leiden
" Leipzig
" Liege
" Lund
" Marburg
" Paris
" Rostock
" Utrecht
" Würzburg

During the past year we have heard from all of the above. 1149 dissertations and 1487 theses have been added to our collection.

We have received in cash from the sale of duplicates for the current year, $159.05.

We have distributed books and journals on exchange account to the following:

Academy of Medicine of Toronto, Canada
American University of Beirut, Syria
Barlow Medical Library, Los Angeles, California
Boston Medical Library, Boston, Massachusetts
College of Physicians and Surgeons, New York City
Harvard University School of Medicine, Boston, Massachusetts
Jefferson County and University of Louisville, Louisville, Kentucky
Medical Library Service, Madison, Wisconsin
Medical Society of the County of Kings, Brooklyn, New York
Milwaukee Academy of Medicine, Milwaukee, Wisconsin
New York Academy of Medicine, New York City
Norfolk County Medical Society, Norfolk, Virginia
St. Louis Medical Society Library, St. Louis, Missouri
Tufts College Medical Library, Boston, Massachusetts
University of Illinois, College of Medicine, Chicago, Illinois
University of Oregon, Medical School, Portland, Oregon
University of Texas, Medical Department, Galveston, Texas
Washington University, School of Medicine, St. Louis, Missouri

We have received exchanges from:

Academy of Medicine of New Jersey, Newark, New Jersey
Academy of Medicine of Toronto, Toronto, Canada
Boston Medical Library, Boston, Massachusetts
Columbia University, New York City
Cornell University Medical College Library, New York City
Harvard University School of Medicine, Boston, Massachusetts
Indiana University School of Medicine, Indianapolis, Indiana
Mayo Clinic, Rochester, Minnesota
Medical College of the State of South Carolina, Charleston, S. C.
Medical College of Virginia, Richmond, Virginia
Medical Society of the County of Kings, Brooklyn, New York
Milwaukee Academy of Medicine, Milwaukee, Wisconsin
New York Academy of Medicine, New York City
New York Homœopathic Medical College and Flower Hospital, New York City
Orleans Parish Medical Society, New Orleans, Louisiana
Queen's University Library, Kingston, Ontario
St. Louis Medical Society Library, St. Louis, Missouri
St. Louis University, School of Medicine, St. Louis, Missouri
St. Luke's Hospital Medical Library, Chicago, Illinois
Tulane University School of Medicine Library, New Orleans, Louisiana
University of Pennsylvania School of Medicine, Philadelphia

University of Rochester School of Medicine and Dentistry, Rochester, New York
University of Texas Medical Department, Galveston, Texas
Vanderbilt School of Medicine Library, Nashville, Tennessee
White Memorial Hospital Library, Los Angeles, California

Several of the exchanges on the above list were made through the Medical Library Association.

With the aid from exchanges and purchases made with the funds appropriated by the Library Committee, we have been able to complete the files of the following journals:

Annales d'Hygiene Publique Industrielle et Sociale
Archiv für Frauenkunde
Archiv für orthopädische und Unfall-Chirurgie
Archives de Maladies du Coeur
British Journal of Radiology
Bulletin de l'Institut Pasteur
Bulletin de la Société Française d'Histoire de la Médecine
Centralblatt für Bakteriologie und Parasitenkunde. (Referate.)
Current Researches in Anesthesia and Analgesia
Dermatologische Wochenschrift
Dermatologisches Centralblatt
Giornale di Batteriologia e Immunologia
Guy's Hospital Gazette
Hygienische Rundschau
Journal of Bacteriology
King's College Hospital Reports
Kongresszentralblatt für die Gesamte Innere Medizin
Long Island Medical Journal
Medical Journal of Australia
Medical Times, New York
Minerva Zeitschrift
Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie
Mittheilungen der Medicinisch-chirurg. Gesellschaft des Kantons Bern
Monatsberichte für Urologie
Monatsschrift für Ohrenheilkunde
The Nurse
Proceedings of the Academy of Natural Sciences of Philadelphia
Therapeutisches Jahrbuch
Tuberculosis
Wiener klinische Wochenschrift
Zeitschrift für Hygiene
Zeitschrift für Nahrungsmittel-Untersuchung und Hygiene
Zeitschrift für Physiologie

In response to requests sent out during the year we have received acceptances from 25 Foreign and 19 American publishers and editors

to send their periodical publications as issued, either as a gift or in exchange for the Transactions of the College. Further we received information that more than 60 American and Foreign journals had ceased publication.

Amount of fines collected for the current year ending November 1, 1930, $55.30.

List of rare medical books and works of special interest received during the year. (These rare books have been acquired without in any way interfering with the purchase of current medical publications):

INCUNABULA

[Total number of incunabula at this date, 391 Titles, 395 Volumes.]

Brack, Wenceslaus. Vocabularium Archonium nuncupatum. [Augustae Vindelicorum, Antonius Sorg, Civis Augustensis], 1487. [Hain No. 3703.]

Fund for Rare Books.

Brutus Novocomensis, Iacobus. Corona aurea de laudibus litterarum.*** Venetiis, Johannes de Cereto de Tridino al. Tacuinus, 1496. [Hain No. 4026.]

Fund for Rare Books.

Gerson, Johannes. Cancellar. Paris. De perfectione cordis. [Parisiis, Felix Balligaut, 1500.] [Copinger No. 2704.]

Presented by Charles W. Burr, M.D., and Edward B. Krumbhaar, M.D.

Johannes. Junior, Ordin. Praedicator. Scala coeli. Vlm, Johannes Zeiner de Reutlingen, 1480. [Hain No. 9406.]

Fund for Rare Books.

Perottus, Nicolaus episc. Sypontinus, Cornucopiae linguae latinae. (Editus a Joanne Pompeio Coriniano et Polydoro Vergilio.) Commentariolus in prooemium C. Plinii. Venetiis, Joannes Tacuinus de Tridino, 1496. [Hain No. 12704.]

Fund for Rare Books.

Philelphus, Franciscus. Orationes. Venetiis, Philippus de Caneto, Mantuanus, 1496. [Hain No. 12925.]

Fund for Rare Books.

Works of Special Interest

Barrère, Pierre. Observations anatomiques, tirées des ouvertures d'un grand nombre de cadavres, propres à découvrir les causes des maladies et leurs remèdes. Nouvelle édition. Perpignan, Reynier, 1753.

Presented by J. Ewing Mears Fund.

Berrettinus, Petrus. Tabulae anatomicae*** opus*** iconas expunxit, perpetuas explicationes adjecit Franciscus Petraglia. Romae, Imp. Venantii Monaldini bibliopolae, 1788. Text and atlas.

Fund for Rare Books.

Boehmer, Philippus Adolphus. Institutiones osteologicae in usum praelectionum academicarum cum iconibus anatomicis. Halae Magd., in off. libraria Rengeriana, 1751.

Fund for Rare Books.

Charleton, Gualterus. Exercitationes de oeconomia animali novis, in medicinia hypothesibus superstructa et mechanicè explicata.*** Hagae-Comitum, Leers, 1681. [Contains also—Cole, W. De secretione animali cogitata. 1681.]

Fund for Rare Books.

Connor, Bernardus. Dissertationes medico-physicae. De antris lethiferis. De montis Vesuvii incendio. De stupendo ossium coalitu. De immani hypogastrii sarcomate. Oxonii, e Theatro Sheldoniano, sumptibus Henrici Clements, 1695.

Fund for Rare Books.

Courtial, Jean Joseph. Nouvelles observations anatomiques sur les os, sur leurs maladies extraordinaires, et sur quelques autres sujets. Paris, d'Houry, 1705.

Fund for Rare Books.

Daran, Jacques. Observationes chirurgicales; sur les maladies de l'urethre, traitées suivant une nouvelle methode. Nouvelle édition. Paris, Debure, 1748.

Fund for Rare Books.

Eustachius, Bartholomaeus. Tabulae anatomicae, quas e tenebris tandem vindicatas et Clementis Papae XI munificentia dono acceptas, praefatione, ac notis illustravit Joh. Maria Lancisius, archiater pontificius. Editio romana altera. Romae, Sumptibus Laurentii, et Thomas Pagliarini, bibliopol. sub signo Palladis, 1728.

Fund for Rare Books.

de Furno, Vitalis. Pro conservanda sanitate, tuendaque prospera valetudine, ad totius humani corporis morbos et aegritudines, salutarium remediorum, curationumque liber utiliss.*** Moguntiae [Schoeffer], 1531.

Fund for Rare Books.

Gelée, Théophile. L'anatomie françoise, recveillie des meilleurs autheurs qui ont écrit de cette science. Lyon, Carteron, 1673.

Fund for Rare Books.

Goelicke, Andreas Ottomar. Historiae medicinae universalis. 2 v. Francofurti ad Viadrum, apud Johannem Godofredum Conradi, 1718–1721.
Fund for Rare Books.

Harvey, William. Exercitationes anatomicae, du motu cordis et sanguinis circulatione.*** Roterdami, Leers, 1654. [Contains also—de Back, J. Dissertatio de corde.*** 1654.]
Fund for Rare Books.

van Meekeren, Job. Rare und wunderbare chyrurgisch- und geneesskünstige Anmerckungen. Nürnberg, Fürsten, 1675.
Fund for Rare Books.

Nollet, [Jean Antoine]. Lettres sur l'électricité.*** Paris, Guerin, 1753.
Fund for Rare Books.

Nuck, Antonius. De ductu salivali novo, saliva, ductibus oculorum aquosis, et humore oculi aqueo. Lugduni Batavorum, apud Petrum vander Aa, 1685.
Presented by S. Lewis Ziegler Estate.

d'Onofrio, Arcangelo. Dettaglio istorico della peste di Noja, in provincia di Bari, suiluppatasi a novembre del 1815. Con un appendice sulla petecchiale corrente. Napoli, Presso Antonio Garruccio, 1817.
Contains also his—Riflessioni teorico-pratiche sulla corrente malattia petecchiale.*** 1817.
Fund for Rare Books.

Pallucci, [Natalis Guiseppe]. Methode d'abbattre la cataracte. Dédiée à Madame la Princesse de Conty. Paris, d'Houry, 1752.
Presented by S. Lewis Ziegler, M.D.

Paracelsus, Aureolus Philippus Theophrastus Bombastus ab Hohenheim Operum latinum redditorum. 2 v. Basilae, ex officina Petri Pernae, 1575.
Fund for Rare Books.

Paracelsus, Aureolus Philippus Theophrastus Bombastus ab Hohenheim Prognosticatio. [Augustae Vindelicorum, per Henricum Steyner], 1536.
Fund for Rare Books.

Plempius, Vopiscus Fortunatus. Ophthalmographia, sive tractatio de oculi fabrica, actione, et usu praeter vulgatas hactenus philosophorum ac medicorum opiniones. Amsterdami, Sumptibus Henrici Laurentii, Anno 1632.
Fund for Rare Books.

Plumptre, James. The plague stayed; a scriptural view of pestilence particularly of that dreadful pestilence the smallpox.*** Cambridge, Hodson, 1805.

Fund for Rare Books.

Priestley, Joseph. History and present state of discoveries relating to vision, light, and colours. London, Johnson, 1772.

Fund for Rare Books.

Purmann, Mathaeus Gottfried. Der rechte und wahrhafftige Feldscher; oder, Die rechte und wahrhafftige Feldschers-Kunst.*** Franckfurt, Rohrlach, 1708.

[Contains also his—Der aufrichtige und erfahrne Pest-Barbierer.***]

Fund for Rare Books.

Ridley, Hum[phrey]. Observationes quaedam medico-praticae et physiologicae; inter quas aliquanto fusius agitur. De asthmate et hydrophobia.*** Lugduni Batavorum, apud Gysbertum Langerak et Theodorum Lucht, 1738.

Fund for Rare Books.

Riolan, Jean. Methodus medendi, tam generalis quam particulariis. Parisiis, Ex off., Plantiniana, apud Hadrianum Perier, 1598.

Fund for Rare Books.

Risueno d'Amador, [Benigno Juan Isiadoro]. Influence de l'anatomie pathologique sur la médecine depuis Morgagni jusqu' à nos jours. Paris, 1836. [Reprint—Mém. Acad. de méd., 1836, v. 6.]

Fund for Rare Books.

Roland, M[arian]. Le cadet d'apollon nay, novrry, et eslevé, sur les ramparts de la fameuse citadelle de Metz, pendant la contagion de l'année passée. 1625.*** Vic, Felix, 1626.

Fund for Rare Books.

Saillant, [Carolus Jacobus]. Tableau historique et raisonné des épidémies catharrales, vulgairement dites la grippe; depuis 1510 jusques et y compris celle de 1780.*** Paris, Didot, 1780.

Fund for Rare Books.

Sanctorius. Sanctorius Medicina statica; or, rules of health, in eight sections of aphorisms. Originally written by Sanctorius, chief professor of physick at Padua. English'd by F. D. London, Starkey, 1676.

Fund for Rare Books.

Sennert, Daniel. Compendium institutionum medicarum.*** Patavii, Imp. Casp. Crivellarii, 1630.

Fund for Rare Books.

Sherley, Thomas. A philosophical essay; declaring the probable causes, whence stones are produced in the greater world. From which occasion is taken to search into the origin of all bodies, discovering them to proceed from water, and seeds.*** London, Cadman, 1672.
Fund for Rare Books.

Sicard, Roch Ambroise. Cours d'instruction d'un sourd-muet de naissance, pour servir à l'éducation des sourds-muets, et qui peut être utile à celle de ceux qui entendent et qui parlent. Paris, le Clere, an VIII [1800].
Fund for Rare Books.

van Slonenbergh, Jasper Hans. Useful transactions for the months of May, June, July, August and September. 1709.*** Translated into English from the Dutch. London, Lintett, 1709.
Fund for Rare Books.

Smellie, [William]. Traité de la theorie et pratique des accouchemens. Trad. d l'Anglois par L. de Preville. Auquel on a joint le secret de Roonhuisen, dans l'Art d'accoucher, trad. du Hollandois. 4 v. Paris, Barrois, 1771.
Fund for Rare Books.

Stubbe, Henry. Indian nectar; or a discourse concerning chocolata.*** London, Crook, 1662.
Fund for Rare Books.

Suetonius Tranquillus, Gaius. Commentationes conditae a Philippo Beroaldo in Suetoniū Tranquillum. Additis q̃ plurimis annotamentis: quae ut facilius cognoscantur.*** Venetiis, Philippus Pincius Mantuanus, 1510.
Fund for Rare Books.

Thacher, James. Military journal during the American revolutionary war, from 1775 to 1783; describing interesting events and transactions of this period; with numerous historical facts and anecdotes, from the original manuscript.*** 2 ed. Boston, Cotton, 1827.
Special Fund for Completing Files of Journals.

Turini, Andreas. Hippocratis et Galeni defensio Hieronymum Fracastorium, de causis dierum criticorum. Romae, Vico Peregrini apud Balthasarem de Cartulariis Perusinum, 1542.
Fund for Rare Books.

Vidius, Vidus. De curatione generatim pars prima. In qua rerum praeternaturam ad humanum corpus pertinentium cognitio, ac curatio in uniuersum methodo exquisitissima pertractantur.*** Florentiae, Apud Bartholomeum Sermartellium, 1587.
Fund for Rare Books.

Manuscripts

Taylor, John. Photostat reproduction of autograph account of the malady of one of his patients, Russian Princess. The original of this is believed to be the only manuscript of Chevalier John Taylor, oculist of 18th century, in existence. In 1893 was presented to the Royal College of Surgeons of England by Thomas Madden Stone.
The photostat copies were made under the direction of Sir D'Arcy Power.

Presented by Burton Chance, M.D.

Other Interesting Additions

A Replica of the Bronze Sedgwick Memorial Medal, awarded to Dr. Mazyck P. Ravenel by the American Public Health Association for Distinguished Service in Public Health.

Presented by Mazyck P. Ravenel, M.D.

We have also received during the past year a number of interesting and more or less valuable autograph letters.

Use of the Photostat: During the past year there have been furnished to Fellows of the College 394 prints, and to other than Fellows, on application, 167 prints. These outside applications include requests from Baltimore, Brooklyn, Cincinnati, Denver, Los Angeles, Madison, Wis., Nashville and Philadelphia.

The work for the Library has been continued as heretofore according to the instructions of the Library Committee, 4379 large and small prints have been made; 10 volumes of incunabula completed, as well as various portraits and manuscripts.

Up to the present time there have been made photostat reproductions of 283 incunabula.

There has been an increase of $5000 to the principal of the Library Endowment Funds through the bequest of Dr. S. Lewis Ziegler, to be known as the S. Lewis Ziegler Library Fund.

A complete list of all the Funds and the expenditures for the year is given in a previous part of this report. All additional expenses, salaries, binding, etc., have to be governed according to the funds appropriated by the College to the Library Committee annually.

An account of stock has been taken and any displacements found arranged in their proper order; no report has been made of any books missing from the shelves.

During the meeting of the American Surgical Association, held in this building May 15–17, 1930, at the suggestion of the Honorary Librarian, an exhibit was made of books or articles written or edited by forty-one original members of the Association. The work of thirty-seven of these original members was represented in this exhibit, together with copies of photographs of twenty-two of the authors taken about the period of 1880.

In connection with the celebration of the College, October 23, 1930, of the Eighteen hundredth Anniversary of the birth of Galen there was an exhibition of some of his works, taken from the collection in the Library of 21 collected works and 93 single works, together with copies of his portrait at different periods.

Library Committee
George W. Norris, M.D.,
Chairman.

INDEX

A

Adler, F. H., discussion on roentgentherapy in pituitary tumors, 226
Aiken, Thomas G., compound fracture of malar bone with involvement of maxillary sinus, 267
Alexander, Franz, psychosis, neurosis and character-disturbance (Nathan Lewis Hatfield lecture X), 96
Amytal, effect of, upon uterus, 47
    sodium, anesthesia, effect of, upon uterus, and its use in obstetrics and gynecology, 47
Anders, James M., discussion on hepatitis, 117
Anesthesia, sodium amytal, additional observations on effect of, upon uterus, and its use in obstetrics and gynecology, 47
Annual report of library committee for, 1930, 277
Appendix, 185
Appleman, Leighton F., cholesterin in anterior chamber, 194
    method of uncovering latent hyperphoria, 190, 193

B

Babbitt, James A., and Fitz-Hugh, Thomas, Jr., agranulocytic angina, 251, 254
Baer, B. F., discussion on modified Le Grange operation for glaucoma, 222
Baer, Louis, discussion on acute mastoiditis with basilar meningitis, 266
Barlow, A., ephedrine and glaucoma, 303
Bologna University, origin of, 173
Bower, John O., discussion of effect of amytal upon uterus, 53
Burr, Charles W., Galen, 155
Butler, Ralph, discussion on specific prophylaxis of pneumococcus and streptococcus meningitis, 262

C

Campbell, Edward H., observations on transillumination of frontal sinuses, 231, 232
Carpenter, John T., discussion on uncovering latent hyperphoria, 192
Chance, Burton, discussion on cholesterin in anterior chamber, 196
    on color-blindness, 214
    on heterochromic cyclitis, 198
    on hole at macula, 200
    on ophthalmic operations, 213
    on tendon transplantation for paralysis of superior rectus, 205

Chance, Burton, Galen's ophthalmology, 166
glioma, 194
skin-grafting eyelids, 193
Coates, George M., discussion on practical points in use of electrosurgical measures in upper respiratory tract, 235
on specific prophylaxis of pneumococcus and streptococcus meningitis, 262
on stricture of oral pharynx, 264
and Ersner, Matthew S., regeneration of mucous membrane of frontal sinuses, 256, 257
Congenital insufficiency of palate, 19
Cowan, Alfred, discussion on bullous keratitis, 187
on cholesterin in anterior chamber, 196
on heterochromic cyclitis, 197
on sclerocorneal trephining in chronic simple glaucoma, 211
incorporation in prescription of style and shape of lenses, 219
Crampton, George S., discussion on incorporation in prescription of size and shape of lenses, 221
Cross, G. H., discussion on modified Le Grange operation for glaucoma, 223
on treatment of vernal conjunctivitis by radium, 200
foreign body in lens, 207
hole at macula, 198, 200

D

Davies, John R., orbital abscess following sinus disease, 264, 265
Davis, Warren B., discussion on transillumination of frontal sinuses, 232
Deaver, John B., hepatitis, 110
Dorrance, George M., bursa pharyngea, 254, 255
congenital insufficiency of palate, 19
treatment of stricture of oral pharynx, 263, 264
Dust, inorganic and pulmonary disease, relationship between, 55
Dustiness, safe limits of, and how they are measured, 66

E

Emerson, Haven, résumé of special inquiry of eye health in Philadelphia, 215
Ersner, Matthew S., discussion on agranulocytic angina, 254

F

Fetterolf, George, discussion on orbital abscess following sinus disease, 265
Fewell, A. G., discussion on uncovering latent hyperphoria, 192
Franchini, Giuseppe, origin of University of Bologna, Italy, 173
Frazier, Charles H., discussion on ophthalmological aspects of pituitary tumors, 230
surgical treatment of pituitary tumor, 227
Fry, W. E., congenital total color-blindness, 213
variations of intraneural course of central vein of retina, 207

G

Galen, 155
Galen's ophthalmology, 166
Gibbon, John H., annual address of President, 1
Gleason, E. B., discussion on specific prophylaxis of pneumococcus and streptococcus meningitis, 262
Goiter and hyperthyroidism, management of, 133
in Philadelphia district, 12
Grant, Francis C., discussion on brain abscess, 237
on specific prophylaxis of pneumococcus and streptococcus meningitis, 261
Grenfell, Sir Wilfred, medicine in sub-arctic (Mary Scott Newbold lecture XXII), 73
Griscom, J. Milton, discussion on bullous keratitis, 188
on skin-grafting of eyelids, 193
modified Le Grange operation for glaucoma, 221

H

Hammett, Frederick S., regeneration reaction to sulfhydryl and its partially oxidized derivatives, 108
Hathaway, Mrs. Winifred, discussion on résumé of special inquiry of eye health in Philadelphia, 217
Holloway, T. B., case of electric cataract, 189
discussion on ophthalmic operations, 213
on sclerocorneal trephining in chronic simple glaucoma, 211
on uncovering latent hyperphoria, 192
on variations on intraneural course of central vein of retina, 208
ophthalmological aspects of pituitary tumors, 229
Heed, Charles R., discussion on foreign body in lens, 207
Hepatitis, 110
Herman, Maxwell, discussion on trachoma in wrestlers, 219
Hewson, William, acute mastoiditis with basilar meningitis, 265, 267
Hirst, John C., and Lapham, Maxwell E., additional observations of effect of sodium amytal anesthesia upon uterus, and its use in obstetrics and gynecology, 47.
Hunter, Robert J., discussion on agranulocytic angina, 253
Hyperthyroidism, goiter and, management of, 133

I

Inorganic dust and pulmonary disease, relationship between, 53
Iridectomy in bullous keratitis, 181

K

Kern, Richard A., discussion on vasomotor rhinitis, 241
Kolmer, John A., bacteriologic and immunologic aspects of iritis, with special reference to its relationship to bacterial allergy, 223

Kolmer, John A., present status of intracarotid method of treatment of pneumococcus and streptococcus meningitis, 258
and Amano, Dr., specific prophylaxis of pneumococcus and streptococcus meningitis, 259, 261, 262

L

Lahey, Frank H., management of goiter and hyperthyroidism, 133
Landis, H. R. M., discussion on relationship between inorganic dust and pulmonary disease, 165
Langdon, H. Maxwell, case of acute vitreous haze following the external use of adrenalin and scopolamin for the separation of synechiæ, 209
discussion on differential diagnosis between tumor and choroidal detachment, 205
on heterochromic cyclitis, 198
on sclerocorneal trephining in chronic simple glaucoma, 212
on uncovering latent hyperphoria, 192
Lanza, A. J., relationship between inorganic dust and pulmonary disease, 55
Lehrfeld, Louis, discussion on treatment of vernal conjunctivitis by radium, 202
Lewis, Fielding O., discussion on acute mastoiditis with basilar meningitis, 267
on compound fracture of malar bone with involvement of maxillary sinus, 268
on orbital abscess following sinus disease, 265
on practical points in use of electrosurgical measures in upper respiratory tract, 234
on stricture of oral pharynx, 263
Library committee, 1930 report, 277
Lukens, Robert M., chrondromata of larynx, 268
on relation of sinus disease to pulmonary infection, from standpoint of bronchoscopist, 247

M

Manges, Willis F., on relation of sinus disease to pulmonary infection, from standpoint of roentgenologist, 249
Mann, Dr., discussion on trachoma in wrestlers, 218
McCrae, Thomas, on relation of sinus disease to pulmonary infection from standpoint of internist, 243
Medicine in sub-Arctic, 73
Memoir of Thomas H. Fenton, M.D., lxxi
Henry Morris, M.D., lxxiii
Charles Eucharist de Medicis Sajous, M.D., lxv
Moore, Ronald C., treatment of vernal conjunctivitis by radium, 200, 203
Mullin, William V., on relation of sinus disease to pulmonary infection from the standpoint of rhinologist, 245
Murphy, F. A., discussion on vasomotor rhinitis, 242
Mütter museum of College of Physicians of Philadelphia, 6

O

Olsho, Sidney L., discussion on incorporation in prescription of style and shape of lenses, 219
Ophthalmology, Galen's, 166
section on, 187

P

Palate, congenital insufficiency of, 19
Parker, Frank C., ophthalmic operations, 203
Pepper, O. H. Perry, discussion on agranulocytic angina, 253
Peter, Luther C., discussion on roentgentherapy in pituitary tumors, 226
tendon transplantation for paralysis of superior rectus, 205, 207
Pfahler, G. E., and Spachman, E. W., roentgentherapy in pituitary tumors, 225
President, annual address of, 1
Proceedings of Section on General Medicine, 270
on Medical History, 275
on Ophthalmology, 187
on Otology and Laryngology, 231
on Public Health and Industrial Medicine, 274
Psychosis, neurosis and character-disturbance, 96
Pulmonary disease, relationship between inorganic dust and, 55

R

Randall, B. Alexander, discussion on acute mastoiditis with basilar meningitis, 267
Ravdin, I. S., discussion on effect of amytal upon uterus, 52
Reese, Warren S., exhibition of case of heterochromic cyclitis, 196
Regeneration reaction to sulfhydryl and its partially oxidized derivatives, 108
Ring, G. O., discussion on roentgentherapy in pituitary tumors, 227
Royer, B. Franklin, discussion on résumé of special inquiry of eye health in Philadelphia, 215
Rose, Edward, and Frazier, Charles H., goiter in Philadelphia district, 12

S

Schenck, Harry P., discussion on vasomotor rhinitis, 242
Mütter Museum of College of Physicians of Philadelphia, 6
Schwartzkopf, George, differential diagnosis between tumor and choroidal detachment, 204
deSchweinitz, G. E., discussion on acute vitreous haze following external use of adrenalin and scopolamin for separation of synechiæ, 210
on bullous keratitis, 188
on color-blindness, 214
on hole at macula, 200

deSchweinitz, G. E., discussion on interesting cases of sclerocorneal trephining in chronic simple glaucoma, 211
on modified Le Grange operation for glaucoma, 222
on uncovering latent hyperphoria, 191
Shannon, Charles E. G., discussion on modified Le Grange operation for glaucoma, 223
on tendon transplantation for paralysis of superior rectus, 207
Shumway, Edward A., discussion on bacteriologic and immunologic aspects of iritis, with special reference to its relationship to bacterial allergy, 224
on treatment of vernal conjunctivitis by radium, 201
emergency outfit for ophthalmic examination and treatment, 217
Shuster, Benjamin H., discussion on acute mastoiditis with basilar meningitis, 267
on brain abscess, 237
Skillern, Samuel R., Jr., discussion on practical points in use of electrosurgical measures in upper respiratory tract, 234
on transillumination of frontal sinuses, 232
Smith, Austin T., two cases of brain abscess presenting some interesting features, 235
Smukler, M. E., discussion on trachoma in wrestlers, 218
Smyth, Henry Field, safe limits of atmospheric dustiness and how they are measured, 66
Spaeth, Edward B., adenocarcinoma of conjunctiva, 189
Sponsler, Marshall B., some practical points in use of electrosurgical measures in upper respiratory tract, 233, 235
Stout, Philip, discussion on transillumination of frontal sinuses, 232
on vasomotor rhinitis, 242
Sulfhydryl, regeneration reaction to, and its partially oxidized derivatives, 108

T

Tassman, I. S., treatment of argyrosis, 212
Thorington, J., discussion on orbital abscess following sinus disease, 265
Tobey, Harold G., vasomotor rhinitis, 238, 242
Tucker, Gabriel, new type of tracheotomy tube with valve, 257

W

Weinberg, M., bacteriology, etiology and serotherapy of infections of wounds (traumatoses), especially of gas gangrene (Mary Scott Newbold lecture, XXIV), 118
Wessels, L. C., discussion on résumé of special inquiry of eye health in Philadelphia, 217

Wood, George B., discussion on bursa pharyngea, 255
on compound fracture of malar bone with involvement of maxillary sinus, 268
on practical points in use of electrosurgical measures in upper respiratory tract, 234
on specific prophylaxis of pneumococcus and streptococcus meningitis, 259
Wounds, infections of (traumatoses), especially of gas gangrene, bacteriology, etiology and serotherapy of, 118

Z

Zentmayer, William, discussion on acute vitreous haze following external use of adrenalin and scopolamin for separation of synechiæ, 210
on cholesterin in anterior chamber, 196
on differential diagnosis between tumor and choroidal detachment, 204
on foreign body in lens, 207
on heterochromic cyclitis, 197
on uncovering latent hyperphoria, 191
iridectomy in bullous keratitis, 187, 189
some interesting cases of sclerocorneal trephining in chronic simple glaucoma, 210
trachoma in wrestlers, 217